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CONTRIBUTORS

NOVEMBER ISSUE

JOHN A. BIGLER, M.D., F.A.A.P. Professor and Head of Department of Pediatrics, Northwestern University Medical School; Chief of Staff, Children's Memorial Hospital, Chicago, Illinois.

ROBERT T. BINFORD, Jr., D.M.D. Assistant, University of Alabama School of Dentistry, Birmingham, Alabama.

EMANUEL CHERASKIN, M.D., D.M.D. Associate Professor of Oral Medicine, University of Alabama School of Dentistry, Birmingham, Alabama.

MILTON B. ENGEL, D.D.S., M.S. Research Associate Professor in Orthodontics, University of Illinois College of Dentistry, Chicago, Illinois.

HAROLD L. HARRIS, M.D. Chief of Anesthesiology, St. Francis Hospital, Evanston, Illinois.

HERBERT R. KOBES, M.D., F.A.A.P. Associate Professor of Pediatrics, University of Illinois College of Medicine; Director, Division of Services for Crippled Children of the University of Illinois, Chicago, Illinois.

HERBERT KOEPP-BAKER, PH.D. Professor of Speech Pathology, Department of Otolaryngology, University of Illinois College of Medicine; Director, Cleft Palate Center and Training Program, University of Illinois Professional Colleges, Chicago, Illinois.

EDWARD F. LIS, M.D. Associate Professor of Pediatrics, University of Illinois College of Medicine; Attending Pediatrician and Director, Center for Handicapped Children, Research and Educational Hospitals, Chicago, Illinois.

PAUL K. LOSCH, D.D.S. Associate Professor of Pediatric Dentistry, Harvard School of Dental Medicine; Chief of Dental Department, Children's Medical Center, Boston, Massachusetts.

RALPH E. McDONALD, D.D.S., M.S., F.A.C.D. Associate Professor and Chairman of Pedodontics, Indiana University School of Dentistry, Indianapolis, Indiana.

MAURY MASSLER, D.D.S., M.S. Professor and Head of Department of Pedodontics, University of Illinois College of Dentistry, Chicago, Illinois.

ARTHUR H. PARMELEE, Sr., M.D., F.A.A.P. Clinical Professor of Pediatrics, University of Southern California School of Medicine; Senior Attending Pediatrician, Los Angeles Children's Hospital, Los Angeles, California.

LAWRENCE K. PICKETT, M.D., F.A.A.P. Clinical Associate Professor of Surgery, State University of New York, Upstate Medical Center at Syracuse; Associate Attending Surgeon, Syracuse Memorial and Syracuse University Hospitals, Syracuse, New York.

DANE G. PRUGH, M.D., F.A.A.P. Associate Professor of Psychiatry and Pediatrics, University of Rochester School of Medicine and Dentistry; Chief of Child Psychiatry Division, Department of Psychiatry, Strong Memorial Hospital, Rochester, New York.

SAMUEL PRUZANSKY, D.D.S. Associate Professor of Orthodontics, Illinois University College of Dentistry; Consultant to Cleft Palate Center and Training Program, University of Illinois Professional Colleges; Chief of Dental Service and Orthodontist to the Children's Center and St. Francis Hospital, Evanston, Illinois.

BERNARD Z. RABINOWITCH, D.D.S. Instructor, University of California Postgraduate Extension Division; Formerly Associate Professor of Pedodontics, University of Southern California School of Dentistry; Consultant, Cedars of Lebanon Hospital, Los Angeles, California.

JULIUS B. RICHMOND, M.D., F.A.A.P. Professor and Chairman of Department of Pediatrics, State University of New York, Upstate Medical Center at Syracuse; Attending Physician, Syracuse Memorial Hospital, Syracuse, New York.

HAMILTON B. G. ROBINSON, D.D.S. Associate Dean and Professor of Dentistry (Oral Pathology and Diagnosis), College of Dentistry, and Professor of Oral Pathology, College of Medicine, Ohio State University, Columbus, Ohio.

WARREN R. SISSON, M.D., F.A.A.P. Pediatrician-in-Chief, Forsyth Dental Infirmary for Children, Boston, Massachusetts.

WAYNE B. SLAUGHTER, M.D., D.D.S. Clinical Professor of Surgery, and Chairman, Department of Plastic Surgery, Stritch School of Medicine, Loyola University, Chicago; Clinical Professor of Surgery and Head of Plastic Surgery, University of Wisconsin School of Medicine, Madison; Attending Staff, Orthopedic and Wisconsin General Hospitals, Madison, Wisconsin, and Mercy, St. Vincent's, The Cradle and St. Francis Hospitals, Chicago, Illinois.

DAVID BERKELEY STARK, M.D., M.S. Clinical Assistant Professor of Plastic Surgery, State University of New York, Upstate Medical Center at Syracuse; Assistant Attending Surgeon (Plastic), Syracuse University Hospital; Assistant Surgeon (Plastic), Syracuse Memorial Hospital; Attending Plastic Surgeon, Veterans Administration Hospital, Syracuse, New York.

PAUL F. WEHRLE, M.D. Assistant Professor of Pediatrics, State University of New York, College of Medicine at Syracuse; Assistant Medical Director, Syracuse City Hospital, Syracuse, New York.

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FOREWORD

JULIUS B. RICHMOND, M.D.

Consulting Editor

The mouth is of concern to the pediatrician from a nutritional, social, psychological, cosmetic and general health point of view. In spite of its significance, however, medical—and pediatric—training includes relatively little instruction concerning the mouth. Perhaps this has been due in part to our delegation of most of the responsibility for such care to our dental colleagues. We are indebted to our dental colleagues for their participation in this volume.

Although each of the clinics of this volume deals with a specific aspect of the child's mouth, a theme running throughout is the concern for the total health of the child. Particularly significant is the emphasis on the relationship of the growth and development of the mouth to that of the entire organism. The implications of this newer knowledge for the clinical management of the child with cleft lip and/or palate, with orthodontic problems or other oral lesions are emphasized by various authors.

Symbolically we invest the mouth with great significance from the time of birth. The early period of life is often referred to psychologically as the "oral" period. Psychological difficulties often find expression through altered oral function. Thus thumb and finger sucking, disorders of speech, ticlike movements of tongue and lips, and abnormalities in eating habits are all examples of the association of oral function with psychological development.

The mouth has many relationships to general health. Although we no longer hold to theories of "focal infection," we do recognize the significance of oral infection to general health. Great advances have been realized in the control of dental caries in recent years. The common oral infections are now more clearly classifiable.

Because of the relationship of oral health to general health, it is

helpful for the physician to have a full understanding of the child's mouth. It is with this objective that this volume is presented. The editor wishes to take this opportunity to express to the participants his appreciation for their resourcefulness and breadth of view in the presentations they have submitted. It has also been a pleasure to work with the publishers, who have been most cooperative in planning and producing this volume.

Syracuse Memorial Hospital
Syracuse 10, New York

THE MOUTH OF THE NEWBORN

ARTHUR H. PARMELEE, SR., M.D.

The mouth of the newborn deserves attention for several reasons. Many of its normal characteristics are unique for this age period and in themselves of interest. In addition, careful examination may reveal abnormal conditions such as congenital malformations of varying degrees of severity and importance, and evidence of infection or birth injury. These items will be discussed in the order mentioned.

NORMAL CHARACTERISTICS

Many of these are of interest because they reflect the particular stage of anatomical development at the time of birth. To some extent they also reflect fetal environment and prenatal or natal experiences and are therefore of clinical value. They are unique to this period and are transitory because of the natural processes of growth and postnatal experiences. Knowledge of these normal peculiarities is essential to recognition of the abnormal.

The *lip margins* show a sharp line of demarcation at the junction of the skin and mucous membrane. The somewhat wrinkled and slightly elevated moist, glistening, red to mildly purplish mucosa ends abruptly at its junction with the smooth pink skin which covers the outer third of the visible lip surface. A few days after birth the mucous membrane presents a superficially furrowed appearance, the surface becomes dry, and soon the outer layer of cornified epithelium gradually separates from the underlying mucosa in the form of crusts or plaques. These are known as *sucking calluses*. They are seen mostly on the central portions of the lips. As they are shed, new ones are formed, and the process often persists for a few weeks.

The *alveolar ridges* of the mandible and maxilla vary considerably in their appearance. Usually the compartments for the individual tooth buds can be seen in the gums in regular arrangement. Frequently there is on the free margin a thin, membranous fringelike tissue with a serrated edge extending in some instances as much as 1 mm. above the firm tissue of the alveolar ridge. This persists for only a few weeks. Not infrequently, small, firm, white *epithelial pearls* or *inclusion cysts* are found on or near the gum margins. These may persist for several weeks or a few months, but are eventually shed. Parents frequently mistake them for teeth. A much more common site for these epithelial pearls is the roof of the mouth, where they occur with great frequency. Here they are pinhead-sized or smaller white bodies often in groups arranged on either side of the median raphe at or near the junction of the hard with the soft palate. They persist only a few weeks. In this site they are known as *Epstein's pearls* or *Bohn's pearls*.

Rarely infants are born with teeth. Usually there are two in the position of the lower central incisors; occasionally there is only one. These teeth are, as a rule, loosely attached to the gum margin and often spontaneously shed within a few days after birth. They are *supernumerary teeth*, since in due time the true deciduous teeth erupt as usual. Rarely one or two true, firmly fixed deciduous teeth will be seen at birth.

A varying degree of *malocclusion* is commonly seen in the newborn. This is associated with an asymmetrical appearance of the face and is due to molding caused by intrauterine posture. The head held, for example, in a fixed position of lateral flexion with the shoulder pressed into the fossa below and posterior to the ear causes the mandible to be pushed toward the opposite side. This causes the face to have a lopsided appearance and leaves a deep fossa in the soft tissues of the neck from the shoulder compression and frequently varying degrees of malocclusion of the alveolar ridges. When the mandible is brought in contact with the maxilla on the side of the shoulder pressure, there will be an appreciable gap between the upper and lower alveolar processes on the opposite side. It is more frequent in infants of primiparous than of multiparous mothers. An inadequacy of maternal calcium metabolism and consequent increased pliability of fetal bones is said to increase the probability of its occurrence. Such malocclusion, in some instances, causes mild and temporary interference with nursing, but is of no clinical importance and corrects itself within a few weeks.

The *frenum of the upper lip* (*frenum labialis superior*) is in some instances prominent and thick, and often there is an associated, rather deep notch in the alveolar ridge of the maxilla. This frenum is a continuation of the fibrous median raphe of the hard palate. It needs no treatment.

A *frenulum linguae* is a common finding. It varies greatly in degree and extent of development; it may be merely a thin vilamentous membrane or may be thick and fibrous; also it may extend barely halfway along the under surface of the tongue or the entire length to the tip. Occasionally a heavy fibrous frenulum that extends to the tip of the tongue will interfere with free protrusion of the tongue and produce a visible notch at the tip (*tonguetie*). Only rarely does it interfere with function. Nursing seems sometimes to be difficult for a short time, but always the infant soon learns to compensate for whatever handicap it may be responsible for. Specialists in the field of speech find little evidence to substantiate a common belief that lisping or other defects of enunciation result from tonguetie. Certainly it is not a serious defect and requires no immediate correction. The practice of clipping a frenulum linguae at birth, common in former years is, fortunately, no longer in favor and is definitely contraindicated, since it establishes a pathway for possible bacterial infection. Bilateral infection of the submaxillary salivary glands followed this procedure in one infant under my care. The frenulum can be clipped at a later date (eight to ten months) easily and safely if it seems indicated. Often the parents feel relieved when it is done.

Pterygoid ulcers (*Bednar's aphthae*), seen frequently in the past, are now only occasionally observed. They present as elongated, oval-shaped grayish patches 8 to 10 by 3 to 4 mm. in diameter, on either side of the midline of the hard palate just anterior to its junction with the soft palate. They are superficial traumatic abrasions of the mucous membrane resulting from attempts to clear the mouth of foreign material at the time of birth. A gauze sponge rubbed across the mucous membrane where it is stretched tautly over the pterygoid processes tears off the superficial mucosa, leaving a denuded area over which a grayish necrotic membrane soon forms. These ulcers are almost always benign and heal rapidly, but troublesome infections have been known to result in rare instances.

Trauma to the oral and pharyngeal mucosa may also result from resuscitation procedures such as suction of the pharynx by bulb or catheter when awkwardly done, or by inexpert use of the tracheal catheter.

Petechial hemorrhages are occasionally seen in the palate of a normal newborn within a few hours after birth. These are probably the result of passive congestion during birth and rhexis of the thin-walled capillaries.

A word should be said about *visualization of the pharynx*. Attempts to depress the tongue with a tongue blade result in strong reflex elevation of the tongue, shutting off completely a view of the pharynx. Of course, by forcing the tongue blade farther back, the infant may be

made to gag, but he also struggles and a good visualization is difficult. A good view of the anterior mouth cavity can be obtained without a tongue blade and by patiently waiting for t' e baby to cry, and adequate view of the pharynx can be had without the use of a tongue depressor. Good lighting is essential, and daylight is preferable to artificial light.

CONGENITAL MALFORMATIONS

The most common congenital malformation of the mouth is *cleft palate and cleft lip* (*harelip*). These defects are usually associated, though one may occur without the other. In some instances the cleft of the palate involves only the soft palate. Only the uvula is cleft in some (*bifid uvula*). In others failure of closure may leave a defect only a few millimeters in length in either the hard or soft palate. The fact that these minor defects occur emphasizes the necessity of a thorough examination of the mouth of the newborn to avoid subsequent embarrassment when symptoms call attention to their presence.

Infants with cleft palate usually require special feeding procedures. Gavage feeding is frequently necessary for a while, or dropper feedings may be successfully used. Later, specially constructed nipples can be used. This subject is covered in more detail elsewhere in this symposium.

Micrognathia due to congenital failure of normal development of the mandible may be associated with cleft palate and sometimes other anomalies of the nasopharynx. When this rare anomaly occurs, there is marked malocclusion of the alveolar ridges of mandible and maxilla in their anterior portions; the tongue is too large for the space provided in the underdeveloped mandible and is forced backward into the pharynx, where it obstructs the air passages and interferes with breathing and swallowing. This is known as the *Pierre Robin syndrome*. These infants usually require emergency care to make adequate respiration possible.* This may be done by anchoring the tongue by means of a suture to hold it forward. Various devices have been used to pull the mandible forward.

Micrognathia or hypoplasia of the mandible of various degrees of severity occurs frequently without associated cleft palate. Most of these are not true congenital malformations, but result from intrauterine posture of such a nature that pressure against the chin interferes with development of the mandible. For example, the head may be sharply flexed so that the chin is held firmly against the sternum during the latter weeks of intrauterine life. Usually this condition of hypoplasia is of relatively mild degree and does not seriously interfere with breathing or swallowing. In a few instances it is severe enough to demand

* More detailed discussion of the management of this syndrome is presented on page 1011.

the same emergency treatment as in the case of the Pierre Robin syndrome.

Ranula is a rare condition characterized by a swelling of the tissues in the floor of the mouth which lifts the tongue upward and forward until it fills the mouth and is displaced forward and laterally. Cysts of the sublingual salivary glands are the cause in the majority of reported cases, but a lymphangioma has been found to be causative in some. Aspiration or incision for drainage of the cysts may be necessary as an emergency measure, since respiration may be seriously interfered with. Surgical excision of the cysts is necessary later.

Mucus cysts are occasionally seen on the labial, gingival or buccal mucosa. They are pea-sized or smaller, glistening, thin-walled cysts due to occlusion of the ducts of mucous glands. They are benign and require no treatment. They usually disappear spontaneously within a few months.

INFECTIONS

The most common infection of the mouth is *thrush*. It is caused by a yeastlike fungus, *Candida* (*Monilia*) *albicans*. Clinically, it manifests itself by the presence of numerous small patches of dull white exudate on the tongue and mucous membranes of the oral cavity. They resemble fine flocculent milk curds, but cling tenaciously to the mucosa and cannot be easily dislodged. The buccal, labial and gingival mucosae and the tongue and palate are often simultaneously involved, but extension to the pharynx is not uncommon. Involvement of the trachea and bronchi and even of the lungs has been noted in rare instances. In cases of pulmonary extension there is evidence to indicate that secondary infections with pyogenic bacteria may be largely responsible for the pneumonitis, at least in some instances.

The presence of thrush is usually not noted until the end of the first week or later. The *symptoms* are mild in most instances or may be lacking entirely. The infant may show some difficulty in nursing and be irritable, but otherwise seem normal. The clinical course is usually short and uneventful when properly treated. Even without treatment the infection tends to clear spontaneously in many instances, but the course is longer.

Treatment. The active treatment of the usual case of thrush is relatively simple. A popular form of treatment in recent years has been application of a 1 per cent aqueous solution of gentian violet by means of a cotton swab to the affected areas of the mouth one or more times daily. Probably one application daily is preferable, since the medication has a drying effect and too vigorous treatment injures the mucosa. This interferes with healing and has on occasion led to extension of

the fungus infection. Also the danger of secondary invasion with pyogenic bacteria is increased. A popular method of treatment before gentian violet came into vogue was the application of a 25 per cent solution of sodium biborate (Borax) in glycerin. This has been so completely satisfactory in my hands that I have never used any other form of medication. A pledget of absorbent cotton saturated with the solution is gently applied to the mucous membranes of the mouth three to four times daily. It is seldom necessary to continue treatment more than 48 or 72 hours.

Recently fungistatic antibiotic preparations have been available. I have had no experience with them.

Control of an epidemic of thrush once it gets started in a newborn nursery is considerably more difficult than the management of individual cases. If a mother has a vaginal infection with *Candida albicans*, her infant may be infected during its birth or later from its contacts with her. Other infants may be infected through contact with contaminated nipples or hands. Isolation of infected infants and careful scrutiny of every step in nursery technique for a possible break are necessary in order to reveal the source and control the outbreak.

Other infections of the mouth cavity are rare under present methods of newborn care. *Infections of the salivary glands* still are seen occasionally.* Direct extension from the mouth by way of the secretory duct is the probable cause. Either the parotid or the submaxillary glands may be involved. The infection is usually unilateral, but may be bilateral, especially in the case of the submaxillary glands. The symptoms are swelling, redness and induration followed by suppuration in a short time unless recognized early and adequately treated with effective antibiotics. When suppuration occurs, pus may drain through the secretory duct; this drainage may be adequate, or incision of the gland may be necessary. The prognosis under proper treatment is good, but sepsis may occur.

Tooth bud infections are extremely rare. Since this disease does still occur and since the symptoms of onset and clinical course are so uniform that recognition and early institution of therapy ought to be the rule, it deserves brief discussion. There is first swelling, redness and induration of the upper lip on one side; this soon involves the cheek and the tissues around the nose and the eye. Inspection of the mouth reveals a swelling of the alveolar ridge of the maxilla on the affected side. Within 24 hours or less an abscess forms which soon ruptures. Often the crown of a deciduous tooth, usually the first molar, is extracted along with the discharging pus. In most cases there is also purulent drainage from the nose due to free pus in the maxillary sinus draining through its natural opening. These infants are extremely ill as

* See the clinic by Dr. John A. Bigler (p. 933) for more detailed discussion.

a rule, and before effective drug therapy was available the mortality was 50 per cent. Those coming to autopsy showed osteomyelitis of the maxilla, while in those who survived the clinical course was that of chronic osteomyelitis. The infection of the tooth bud is probably primary and invasion of the antrums secondary. Evidence has been presented indicating the possibility of primary infection of the antrum with subsequent infection of the tooth bud. However, the involvement of the tooth bud at the onset of the disease in each case plus the fact that infections of tooth buds of the lower jaw have also been reported makes primary involvement of the tooth bud seem much more probable.

BIRTH INJURIES

The newborn seldom sustains serious direct injury to the mouth during birth. But injury to peripheral nerves or the brain may disturb its appearance and its functions.

Facial paralysis due to injury of the seventh nerve causes distortion of the mouth and interferes with sucking. Fortunately this paralysis is almost always of short duration and the infant's health does not suffer from it. In the rare cases of permanent facial paralysis asymmetry, malocclusion, drooling and interference with swallowing and with speech result.

Paralysis of the vocal cords and, in rare instances, of the pharyngeal muscles results from birth injury to the recurrent laryngeal nerve. Aphonia and/or difficulty in swallowing are the symptoms. When the gag reflex is impaired, there is danger of aspiration pneumonia. Here too the paralysis is usually transitory, but since it may persist for two or three weeks, the consequences associated with aspiration may be serious.

Paralysis due to brain injury or congenital defects of the brain rarely affects the mouth. However, paralysis of the pharyngeal muscles may in rare instances result from brain injury at birth. In such cases the paralysis is usually permanent.

Severe and persistent trismus due to brain injury or brain defect is another rare condition. I have seen only a few instances, and in each it was associated with other evidences of severe brain damage. Autopsies performed on three of these showed intraventricular hemorrhage in two and severe hydrocephalus in one.

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4614 Sunset Boulevard
Los Angeles 27, California

EXAMINATION OF THE MOUTH

RALPH E. MC DONALD, D.D.S.

It is not uncommon for the physician to be the first one to have an opportunity to examine the child's mouth. Though the dentist recommends that the child be examined at least by the age of two and a half to three years, often the parents do not follow this advice, and unless some abnormality is noticed, or the child has an accident, the first dental appointment is often delayed.

Dental caries and many oral anomalies are evident early in the child's life, and the physician in his routine oral examination of the child can often detect deviations from normal and refer the child to the dentist for treatment.

In the examination of the child's mouth the soft tissues, particularly the gingival tissues, should be carefully observed, since they often show the first deviation from oral or systemic health of the child.

GINGIVITIS

It has been estimated that approximately 30 per cent of young adults have diseases of the periodontium which started during childhood or adolescence. Though it is true that a severe gingivitis is rather rare in children, except when it accompanies a systemic disturbance, a mild type of gingivitis or inflammation of the superficial part of the periodontal tissue which surrounds the cervical portion of the teeth is common. Though it is seen so frequently that many minimize its significance, the physician and dentist should constantly look for this type of gingivitis in the routine examination and attempt to classify the abnormality as to cause, type and treatment.

From the Department of Pedodontics, Indiana University School of Dentistry, Indianapolis.

The gingivitis most often seen in the child is caused by a lack of oral hygiene and by the accumulation of food debris, calcareous deposits or other local traumatic and toxic agents between and around the necks of the teeth. Since it is usually a mild progressive inflammation and there is seldom any pain associated with it, there is a tendency for the patient to ignore it. The inflammation is usually confined to isolated areas of the mouth and affects only the free margin of the gingiva. If the local irritants are not removed and a proper toothbrushing regimen followed, the underlying alveolar bone may begin to break down and a periodontitis may result. The degree of severity of the gingivitis is no doubt related to the general health of the child and influences the general destruction of the supporting tissues.

VINCENT'S INFECTION (ACUTE NECROTIZING GINGIVITIS)

This has been described as an acute inflammatory disease or infection of the gingival borders, the crest of the interdental papilla and

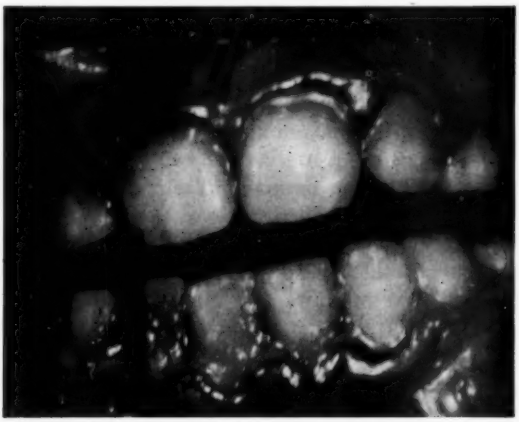

Fig. 1. Oral Vincent's infection in which a gray necrotic membrane is evident on the margin of the enlarged, painful interdental papilla. Vincent's infection in the child is seldom seen and should not be hastily diagnosed.

sometimes the tonsils and palate. Though it is frequently seen in young adults, it is rarely seen in children. It is characterized by the formation of a gray necrotic membrane and small ulcers which are localized upon a painful hyperemic gingiva (Fig. 1). The necrotic membrane may be easily removed, leaving an exposed, raw surface. The temperature frequently will rise to 102° F. or above, and the child will experience general malaise. There is a characteristic fetid odor. The acute stage of the infection responds dramatically, usually within 48 hours, to a

thorough cleansing of the mouth and the use of oxidizing sprays and mouth rinses.

It cannot be emphasized too strongly that Vincent's infection is extremely rare in the child. In the author's experience it has never been observed in the preschool child and only occasionally in a child under nine years of age. One should guard against making a hasty diagnosis of Vincent's infection. Occasionally what first appears to be Vincent's is, instead, an oral manifestation of a more severe systemic disturbance. Not infrequently oral changes associated with Hand-Schüller-Christian and Letterer-Siwe disease have, in the early stages, been inadvertently diagnosed as Vincent's infection.

ACUTE HERPETIC GINGIVOSTOMATITIS

This is a stomatitis usually limited to infants and young children. It is caused by the herpes virus, which attacks the epithelial cells. The clinical course somewhat resembles that of Vincent's infection, and it is frequently incorrectly diagnosed as such.

The gingiva is fiery red and painful and has a tendency to bleed when touched. There is an absence of the gray necrotic membrane characteristic of Vincent's infection, and the mouth may be extremely clean and free of debris.

Rather well defined ulcers, 2 to 3 mm. in diameter, can be seen on the lips, buccal mucous membrane and tongue. The child will run a low grade fever, will be irritable and, because of the tenderness of the mouth, unable to eat.

Again, in contrast to a true Vincent's infection, the herpetic stomatitis will run a 10 to 14 day course. The acute symptoms, however, can be reduced and the course shortened by keeping the mouth clean and by local applications of soluble Aureomycin to the lesions. There is a tendency for recurrence, although subsequent attacks are less severe. The cases do not respond to either topical or intramuscular penicillin therapy. In fact, there is some evidence that penicillin may act as an antagonist actually prolonging the clinical symptoms.

SCORBUTIC GINGIVITIS

Often the first indication of a vitamin C deficiency can be noted by changes in the gingival tissue. This gingivitis differs from the marginal type often associated with poor oral hygiene and calcareous deposits in that there is a tendency to spontaneous hemorrhage. The bleeding which occurs is of a purpuric type and is the result of fragility of the capillaries. Typical scurvy in children is usually seen only before the child is placed on a generalized diet. Cases of subclinical scurvy, how-

ever, exist in patients of all ages, and the differential diagnosis may be difficult.

At the time of the eruption of the primary or even the permanent teeth a swollen area resembling a hematoma may appear before the tooth emerges through the tissue (Fig. 2). This is often interpreted as evidence of vitamin C deficiency. After the eruption of the tooth there may be continued enlargement of the purplish mass, spontaneous hemorrhage and even secondary infection.

Fig. 2. A purplish mass of tissue resembling a hematoma and partially covering the teeth at the time of eruption is evidence of vitamin C deficiency, or scorbutic gingivitis.

In the absence of local irritants and of systemic conditions the scorbutic gingivitis responds quickly to vitamin C therapy.

DILANTIN HYPERPLASIA

Hyperplasia of the gingival tissues is occasionally seen in a child and may be of the inflammatory or fibrous type. Both types of gingival enlargement may result from chronic, local irritation, but the inflammatory type, in particular, may be a manifestation of vitamin C deficiency or leukemia.

The fibrous type may be idiopathic or associated with the use of Dilantin Sodium. In Dilantin hyperplasia the tissues are firm and resilient and present a minutely lobulated appearance. Occasionally the condition may be complicated by secondary inflammatory changes with edema, resulting in an increase in size and a bulging of the tissue away from the tooth surface. As a result of the swelling the surface demarcation of the individual gingival papilla becomes less pronounced.

In some cases the hyperplasia, even when associated with Dilantin therapy, can be controlled by adequate prophylaxis and oral hygiene. Surgical removal, or gingivectomy, is occasionally indicated in severe cases, although there is a tendency for recurrence, particularly without excellent patient cooperation and home care.

PERIAPICAL INFECTION (ALVEOLAR ABSCESS)

In the examination of the child's mouth a careful search should be made for evidence of nonvital teeth. It is not uncommon for one of the primary teeth to become nonvital as a result of deep caries or trauma, without the child's experiencing any pain. Chronic draining fistulas and evidence of nonvital teeth of long standing can be easily overlooked unless the soft tissue opposite the root apices is carefully examined. Areas of localized swelling and redness are usually indicative of periapical infection with a rarefaction of the bone. The involved tooth will usually be slightly loose, and there may be evidence of an exudate or pus escaping from around the tooth. The soft tissues in the early stages of the infection will be swollen and red and painful to touch. The tooth will usually be uncomfortable during mastication even though it has not presented acute symptoms of pain.

The teeth in the area as well as those opposing the involved tooth will be covered with debris, indicating a lack of function. After the acute symptoms have been reduced the tooth should be extracted or occasionally root canal therapy undertaken to remove the possible foci of infection and to aid in the reduction of a localized gingivitis resulting from the lack of function. Eliminating the source of infection will also prevent damage to unerupted permanent teeth as well as other teeth in the area.

It is not uncommon for unerupted permanent teeth that have undergone growth and calcification in an area approximating the alveolar abscess to erupt later, showing evidence of the abnormal local environment. The defect on a permanent tooth may be noted as incomplete coalescence and calcification of pits and fissures. If the area of bone involvement surrounding the developing tooth has been extensive, the crown may show evidence of hypocalcification or hypoplasia and be quickly attacked by dental caries. An illustration of this can be seen in Figure 3. Occasionally a cyst will develop around the unerupted tooth and the tooth germ may be destroyed even before eruption.

The roots of nonvital primary teeth will seldom undergo normal physiologic resorption, but instead may remain in the alveolar bone or soft tissue as foreign bodies to deflect or prevent the eruption of permanent teeth in the area (Fig. 4).

In addition to the dangers just cited, badly decayed and nonvital

teeth serve as incubation areas for microorganisms, and it is impossible to reduce the oral bacterial count until the teeth have been removed or treated and restored. Because of the local environment, sound teeth

Fig. 3.

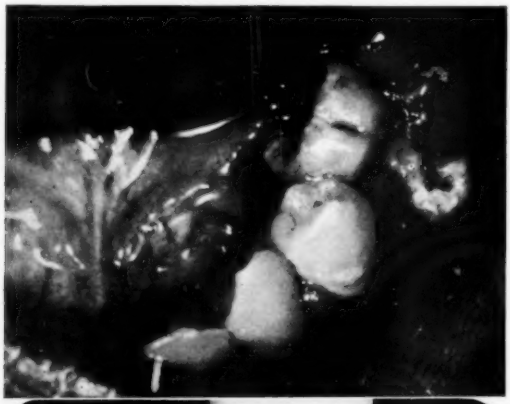

Fig. 4.

Fig. 3. Hypoplasia and malformation of the crowns of permanent teeth are frequently seen when they undergo maturation beneath nonvital primary teeth.

Fig. 4. Radiograph of a nonvital primary tooth. The roots have not undergone normal resorption, and this has resulted in a deflection of the permanent tooth as well as malformation of its crown.

in the area may become carious, which fact is reason enough to recommend the removal of badly broken-down or nonvital teeth.

PROBLEMS RELATED TO THE ERUPTION OF TEETH

The eruption of teeth certainly can be looked upon as a normal physiologic process and only occasionally complicated or altered by

systemic disturbances or local conditions which may influence the pattern. Though it is true that both the primary and permanent teeth follow a fairly predictable eruption pattern, the time of eruption varies greatly with different children. Eruption should not be considered delayed or premature just because the individual child deviates from the "eruption table." A radiographic survey of the dentition is essential in determining whether the child is deviating from a so-called normal eruption time. By making use of the ultra-fast dental film and with the parent holding the child and the film it is possible to take radiographs of the developing dentition of the infant or small child. The radiographic survey is not recommended as a routine procedure before the teeth erupt unless there is reason to suspect some abnormality.

Natal Teeth

It is not too unusual to notice a primary tooth erupted at birth; in fact it is reported to occur at least once in approximately 2000 births. The appearance of a tooth at birth or its eruption during the neonatal period is not usually associated with a systemic disturbance, nor should it be considered a supernumerary tooth unless verified by x-ray film. Instead, the natal tooth should be looked upon as a prematurely erupted tooth and an indication that an early eruption pattern may be expected for the remaining teeth. Natal teeth as well as early eruption may follow a familial pattern, and it is not uncommon to find a history of this early eruption in other members of the family. The prematurely erupted natal tooth has little root formation and little bony support and may be attached only at the neck to the gingival tissue. Even though the tooth is loose, there is little danger of its becoming detached, aspirated or swallowed. Because of its looseness and movement, however, it may be painful or interfere with nursing. Since the tongue lies between the alveolar ridges at birth, the sharp incisal edge of the tooth may lacerate the sublingual area of the tongue.

Unless the tooth is causing some difficulty it should not be removed. If it is necessary to remove the tooth, the pedodontist or oral surgeon will dissect the tooth away from the fragile attachment to prevent tearing of the tissue and excessive hemorrhage.

Difficult Eruption

Some children experience difficulty in erupting the primary teeth. The teeth may be slow in emerging through the tissue, and the gums may be swollen and occasionally inflamed. The inflammation may follow the placement of fingers or unclean objects in the mouth, and a localized infection may develop. Also at the time the tooth emerges

a flap of tissue may partially cover the tooth, with the development of a pocket or a food trap resulting in a localized infection or pericoronitis.

It is possible for a fever to result from an eruption gingivitis or local infection. Systemic reactions such as diarrhea and croup, however, are usually coincidental to the teething process rather than the result of the normal eruption process.

Lancing the gum tissue to aid the eruption of the teeth is rarely indicated, since a secondary infection may follow the surgery and the mouth will be very sore. In some cases of surgical intervention the eruption may be delayed even more because the gum tissue will quickly heal over the area with dense scar tissue. Mild sedation and providing the child with something clean on which to chew will frequently be helpful in carrying the child through the rather brief but irritable periods.

Delayed Eruption

The physician in his oral examination frequently sees evidence of delayed eruption. Tardy or delayed eruption of the primary or permanent dentition by several months should not be looked upon with alarm, provided the eruption is following a symmetrical pattern.

A delay in the initiation of the eruption of primary teeth beyond one year may be related to a systemic or congenital condition. Those conditions most frequently associated with a marked delay in eruption are mongolism, cleidocranial dysostosis, cretinism, rickets and congenital syphilis. When there is delayed eruption of individual teeth, the cause should immediately be determined, since delayed eruption of individual units is often followed by loss of space and malocclusion.

Delayed eruption of one or more permanent teeth frequently results from prolonged *retention* of primary teeth. As was mentioned earlier, nonvital primary teeth may not undergo normal resorption, and the retained root or the tooth may cause deflection or prevent the eruption of the permanent tooth (Fig. 5). Occasionally a traumatic injury to the primary teeth will result in the displacement of the developing permanent tooth germ with the effect that it will follow an abnormal eruption path and perhaps be greatly delayed in its eruption.

The *ankylosis of primary molars* is often responsible for delayed eruption of the permanent successors. Root resorption of the primary molar begins as early as four years of age. The resorption is not a continuous process, but is interrupted by intermittent periods of rest and even repair, in which alveolar bone is laid down into the resorbed lacunae. If the repair is excessive, the continuing resorption process may not overcome the bony union between the dentin of the root and the alveolar bone, and the primary tooth becomes firmly attached or

ankylosed. Growth is then interrupted in this localized area, and the primary tooth fails to erupt and keep pace with the adjacent teeth. Evidence of this can be noted when the teeth are in normal occlusion, since there will be a lack of occlusion and often several millimeters of space between the ankylosed tooth and its normal opponent. If ankylosis persists, the permanent tooth will either fail to erupt or will be deflected from its normal position.

Impaction of the permanent tooth is another frequent cause of delayed eruption. Impaction usually occurs because the tooth has deviated from its normal eruption path. However, some permanent teeth, particularly maxillary permanent cuspids, will be impacted in the

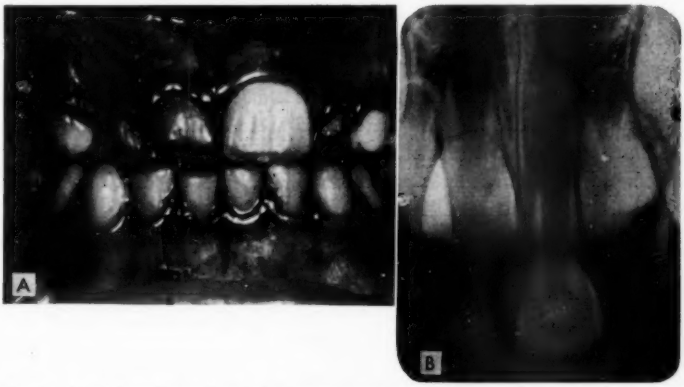

Fig. 5. A, Delayed eruption of a permanent maxillary central incisor due to the prolonged retention of a primary tooth. *B*, Radiograph showing a lack of root resorption of the primary incisor and delayed eruption of the permanent tooth.

palate for no apparent reason. The earlier the impacted tooth is discovered, the greater the chance for recovery or adequate correction.

Supernumerary teeth frequently cause tardy eruption or noneruption of permanent teeth and may be the origin of malocclusion. Supernumerary primary teeth are relatively rare and usually do not cause gross abnormalities in the occlusion. Supernumerary permanent teeth occur with much greater frequency, and surveys indicate that extra teeth occur at least once in every 110 children. They do not occur nearly so frequently as do missing teeth, but their presence, unless discovered early, has a profound influence on the dentition and the ultimate occlusion of the teeth.

The most common site of occurrence of supernumerary teeth is in the maxillary anterior area, although they have been reported in all quadrants of the mouth. They may take the form and size of normal teeth, or they may be small calcified masses barely resembling tooth

form. They may erupt and be first discovered by the child or parent. More frequently, however, they fail to erupt and are undiscovered until an abnormality is noted in the eruption of teeth or the occlusion of the child. Supernumerary teeth in the midline are often referred to as "mesiodens" and may cause impaction, torsiversion, labial displacement of the teeth or the persistence of a wide diastema. If they are inverted in the maxilla, they may erupt and penetrate the floor of the nose. In addition to causing delayed eruption, they occasionally form cysts which may involve the entire palate or alveolar process.

Since supernumerary teeth most often occur in the maxillary midline area, it is not uncommon to observe a late eruption of one or both of the permanent central incisors. Although supernumeraries are usually removed as soon as they are discovered, occasionally the dentist prefers to wait until root development of the adjacent teeth approximates the stage when they will not be damaged during the surgical procedure. If undetected, midline supernumeraries will usually delay eruption of one or more of the anterior teeth, and the adjacent teeth after eruption may drift into the area and take up the space needed for the delayed tooth. Even after the removal of the supernumerary tooth and a reopening of the space, the unerupted tooth may not come into its proper position for months or even years. This is particularly true if root development is complete, in which case the delayed tooth seems to lose much of its eruptive power. Even after eruption they rarely assume a normal position, but instead are usually rotated or are out of alignment. This is reason enough to be aware of the problem and constantly watch for evidence of supernumerary teeth.

Missing Teeth

Approximately 5 per cent of all persons fail to develop some of their teeth, this figure being in addition to the absence of third molars. The lower permanent central incisor is the tooth most frequently missing, followed in frequency by the upper lateral, the second bicuspid and third molars. What is sometimes mistakenly interpreted as delayed eruption may be verified radiographically as the congenital absence of teeth. The most frequent indication of missing teeth, outside of their failure to appear in the mouth, is the presence of a diastema, or spacing of the teeth. While a diastema may be due to a number of factors, such as microdontia, macroglossia, malocclusion or abnormal frenum, supernumerary teeth or even odontomas should be suspected as being responsible for the abnormal spacing.

In cases of missing teeth it is important for the pedodontist or orthodontist to see the child early in order that a long-range treatment plan can be outlined. It is important to determine early whether spaces

resulting from missing teeth should be closed or held open for future prosthetic replacements.

Abnormal Labial Frenum

The question often arises in the mind of the physician concerning what constitutes an abnormal labial frenum and its effect on the alignment of the teeth. The labial frenum frequently takes the form of the wide band of tissue with fibers that pass between the central incisors and attach to the hard palate rather than the labial mucosa. This may occasionally result in spacing of the primary or permanent central incisors. It is safest to delay the diagnosis of an abnormal frenum until after the eruption of the permanent incisors and cuspids. It should be remembered that the maxillary permanent incisors erupt with a slight axial inclination with a space between them, the amount of which varies considerably. This stage of the dentition is often referred to as the "ugly-duckling" stage. The lateral incisors also erupt with a distal inclination, but as they erupt they tend to close the space between the central incisors. With eruption of the cuspids the space is closed more, and this closure often brings about an atrophy or shrinking of the frenum. Only in those cases in which the frenum is in the form of a wide band with an attachment to the lingual side of the alveolar process and in which it persists after eruption of the anterior teeth should the frenum be suspected as being an etiologic factor in causing the spacing of the maxillary permanent incisors. This statement is strengthened by those who believe that the surgical elimination of the frenum has nothing to do with the eventual alignment of the teeth.

Developmental Anomalies of the Teeth

Disturbances in the development of the enamel of the teeth usually fall into one of three classes, those being hereditary with a history of similar conditions in other members of the family, those resulting from a systemic disturbance or nutritional deficiency and those related to an abnormal local environment.

The enamel defect most commonly noted is referred to as *hypoplasia* resulting from a deficiency or disturbance at the time of matrix formation or calcification of the tooth. Enamel hypoplasia of the primary teeth is uncommon and, when it does occur, is usually prenatal in origin. It is often difficult to relate it to or correlate it with other than a vague history of difficulty or abnormal experience during pregnancy. Usually only the occlusal third of the crowns of the primary teeth is involved and only rarely the anterior teeth. The cusps and the

occlusal surfaces may be irregularly formed and not necessarily susceptible to early decay.

In the permanent dentition the incisors and the first permanent molars are the teeth most frequently involved. Occasionally the incisal portion of the cuspid is involved, and only rarely do the bicuspids and second permanent molars show evidence of defective development. The enamel defect may be limited to a smooth surface discoloration, in which case it is referred to as *hypocalcification*. More severe cases may demonstrate pitting of the enamel, or there may be areas actually devoid of an enamel covering. Prolonged fevers or nutritional deficiencies are the most common etiologic factors, and all the teeth undergoing development or maturation at the time of the disturbance will show evidence of the condition (Fig. 6). It is often necessary for the dentist to cover the defective areas to reduce the sensitivity of the exposed dentin, control dental caries or improve the appearance of the teeth.

Fig. 6. Enamel hypoplasia of the permanent anterior teeth. Although it is often difficult to determine the cause, a nutritional deficiency is most often associated with this condition.

Amelogenesis imperfecta, or brown teeth, is often mistaken for enamel hypoplasia. It is a hereditary anomaly, however, following a dominant pattern, and, in contrast to enamel hypoplasia, is seen to affect all the teeth in both the primary and permanent dentitions. The teeth are covered by a thin layer of abnormally formed enamel through which can be seen the dark underlying dentin. Although they are not unusually susceptible to dental caries, it is often advisable to protect the posterior teeth with overlays. As soon as the anterior teeth erupt suffi-

ciently, they may be restored with jacket crowns to improve the appearance.

Dentinogenesis imperfecta, or hereditary opalescent dentin, is another condition often grouped with the hypoplasias. The two should not be confused, however, because, in contrast to hypoplasia, all the teeth, both the primary and permanent, are affected and the defect is limited to the dentin. The odontoblasts fail to differentiate properly, and the result is a brown, poorly calcified, soft dentin. The dentino-enamel junction is atypical, allowing the enamel to break away and leaving the soft underlying dentin exposed, to be rapidly abraded. Often the primary teeth, by age six, are abraded to the level and contour of the alveolar ridge. Early observation by the dentist is essential so that the severe cases can be protected by overlays to prevent the gross destruction of the tooth structure.

EVIDENCE OF ABNORMAL HABITS

Many children develop some type of finger-biting or -sucking (Fig. 7) habit which, if continued over a long time, may result in an abnormal

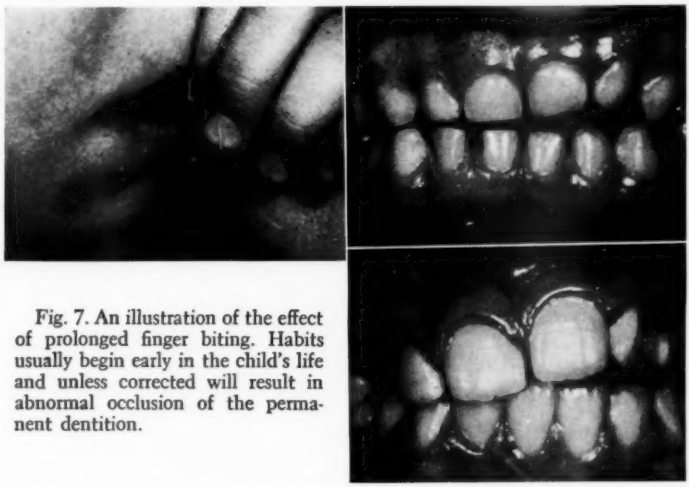

Fig. 7. An illustration of the effect of prolonged finger biting. Habits usually begin early in the child's life and unless corrected will result in abnormal occlusion of the permanent dentition.

occlusion. Thumb sucking is perhaps the most widely practiced habit and one which may have the most profound effect on the dentition. The effect of sucking habits on the maxillary and mandibular bones and the dental arches, including the occlusion of the teeth, depends upon the frequency with which the habit is practiced and the duration of time the habit has lasted. Other factors that are important contributory causes in determining the presence, type and severity of a

malocclusion are the osteogenic development, the genetic endowment of the child and general health.

Contrary to common belief, thumb sucking is not always harmful to the occlusion. In fact, occasionally the occlusion, particularly in cases of prognathism, is helped by the habit. Nevertheless we must always be aware of the potential damage to the denture and the psychological implications of the habit, which, if continued over a period of years, is not socially acceptable. It is perhaps the obligation of the dentist to point out to the parent any existing damage to the occlusion and predict the course should the habit be continued.

Thumb sucking often has its origin in infancy or early childhood and is started as a result of a feeding problem and is then carried on over the years merely as a habit. In other cases the cause is obscure and may be related to a feeling of insecurity, maladjustment or complex problems in the home environment. It is often impossible for the dentist or physician to determine the basic underlying cause of the habit, in which case the child and parent should be referred to specialists in the field of child psychiatry.

If the habit is discovered in the young child before it has been practiced too long, there is a good chance that the resulting defect, if any, will be self-correcting. Some are of the opinion that if thumb sucking is not practiced over four years, there is a strong tendency for self-correction in the transitional dentition.

Private discussions with the older child are often helpful in seeking out the cause of the habit. Often there can be established a working relationship between the child and the dentist which will be helpful. If the child really wants to discontinue the habit, often an appliance placed in the mouth as a reminder will be all that is necessary. A mouth shield worn between the teeth and lips acts as a habit breaker and also as a corrective appliance.

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1121 West Michigan Street
Indianapolis 2, Indiana

THE DIAGNOSIS AND MANAGEMENT OF ORAL INFECTIONS

PAUL F. WEHRLE, M.D.

Oral infections due to various bacterial, viral and mycotic agents are frequently encountered in children. Although some of these infections are self-limited, others require prompt diagnosis and proper therapy for optimal results. A review of these infections seems desirable, particularly since there has been much confusion regarding the proper classification of several of these infections. The present discussion is concerned chiefly with some of the more recent developments concerning their epidemiology, recognition and therapy.

This discussion will be limited to those infections seen most frequently, and is divided into two sections. The first includes those infections either limited to the mouth or producing their main clinical manifestations in this area. The second includes those infectious diseases with the majority of their manifestations apparent in other portions of the body, but with characteristic and even diagnostic lesions located in the mouth.

INFECTIONS WITH PREDOMINANTLY ORAL MANIFESTATIONS

Acute Herpetic Gingivostomatitis (Herpetic Stomatitis, Ulcerative Stomatitis, Trench Mouth or Apthous Stomatitis)

Etiology. There has been considerable confusion regarding the etiology and terminology of this condition. However, it has become increasingly apparent since the isolation of herpes simplex virus from a series of typical cases by Dodd, Johnston and Buddingh[6] that it is the responsible agent. This entity represents perhaps the most common clinical manifestation indicating primary invasion of a susceptible person by herpes simplex virus. The dramatic entry of the virus with

From the Department of Pediatrics, State University of New York, College of Medicine at Syracuse, and the Syracuse City Hospital.

the production of disease is not the usual course of events, however, as might be anticipated from the nearly ideal host-virus relationship seen in many adults with the mild recurrent fever blisters also caused by this same virus. Indeed, the development of acute gingivostomatitis appears to be a rarity when all infections with this agent are considered. Figure 8 illustrates the dynamics of this infection, with some of the variations seen in the oral mucous membrane.

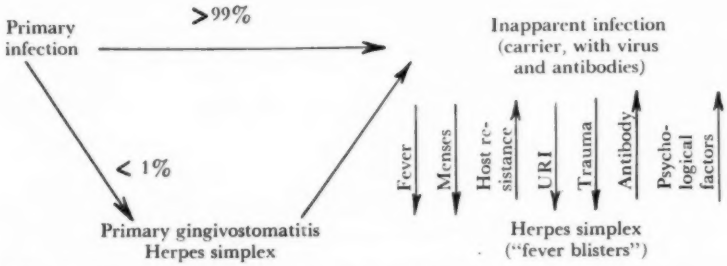

Fig. 8. Oral manifestations of herpes simplex virus. (After Blank and Rake.[2])

As indicated in this diagram, the great majority of the initial infections do not appear to be associated with symptoms or clinical manifestations. Since these infections are entirely silent at the onset, they are classified as inapparent infections. The recognition of the time and occurrence of the infection is possible only by laboratory techniques involving either the isolation of herpes simplex virus or the demonstration of specific antibodies. With the entry of the virus in this clinically inapparent form an exceedingly delicate balance between the host and virus is established, which gives considerable insight into the nearly perfect adaptation of this host to man. With the occurrence of events such as respiratory infections, trauma (sunburn) or emotional disturbances this balance may be tilted to favor the virus with appearance of "fever blisters." It is apparent that the factors influencing this balance vary considerably in different persons. In some persons normal menstruation seems sufficient to allow the occurrence of these lesions, while in others a severe episode of pneumococcal meningitis is required.

A relatively small minority of primary infections, as noted in Figure 8, may present as an acute gingivostomatitis. This manifestation appears chiefly among children, and predominantly among those from one to three years of age. This age distribution of cases parallels the age at which the herpes simplex virus is most frequently first encountered, since studies of virus isolation and antibody acquisition suggest

that most primary (inapparent and clinical) infections occur during the first three to five years of life.[4, 12] This pattern, not unlike that of *Hemophilus influenzae* infection with the relatively rare occurrence of meningitis,[7] serves to emphasize the ubiquity of this virus.

Clinical Manifestations. The usual presenting complaints in acute herpetic gingivostomatitis are fever of 101 to 103° F. or even higher, failure to eat or pain referable to the mouth in older children, abdominal discomfort, malaise and profuse salivation. Indeed, the "drooling" is so marked in many of these children that their wet shirt-fronts may give a clue to the proper diagnosis in the waiting room before examination, since in few conditions is there such profuse salivation together with apparent discomfort on swallowing.

Physical findings include swollen hyperemic gums which bleed on slight trauma and multiple shallow ulcers, most frequently involving the tongue and buccal mucosa, particularly in the gingivobuccal region. The gums, lips and palate may also show areas of ulceration, and at times a cutaneous lesion is also present, as shown in Figures 9 and 10. The ulcers follow the development of vesicles, with subsequent collapse of the roof of the vesicle. These vesicles are no longer present by the time the child is customarily seen. Regional adenopathy may be marked, and foul breath resulting from the necrotic tissue is usually present.

Diagnosis. The clinical laboratory offers little assistance in the diagnosis of this entity, since the white blood cells and differential counts may be variable, and smears and cultures from the oral lesions show mixed flora.[11] The isolation of virus and determination of antibody increase are complex techniques in the province of the virus research laboratory. Fortunately, the appearance of the lesions is so characteristic that clinical diagnosis is entirely satisfactory.

Course. The course of the disease is variable, but symptomatic improvement is usually apparent in four days to one week, and the lesions are usually no longer present 10 days to two weeks after the onset.

Treatment. Therapy is directed toward two goals. These are the maintainance of fluid and nutritional balance and therapy for the local lesions. The first goal may be difficult to achieve, since the physician is usually faced with an irritable young child with an extremely sore mouth. Fluid intake is best maintained by first ascertaining the child's preferences regarding beverages. Those that are bland are selected and offered ad lib. either cold or with ice. Milk, chocolate milk and water often suffice, although more ingenuity is sometimes required. Other foods helpful during the acute stages are Jello, custards, Junket and ice cream. Fruit juices such as orange juice, and coarse-textured or highly seasoned foods may cause discomfort. These are almost invariably rejected and will add considerably to the problem of food accept-

ance if offered persistently. Occasionally parenteral fluids may be required in infants with extensive lesions.

The second problem, that of the local lesions, is of less consequence. There is at present no antibiotic or other agent effective against the herpes simplex virus in man. However, in severe cases with apparent

Fig. 9.

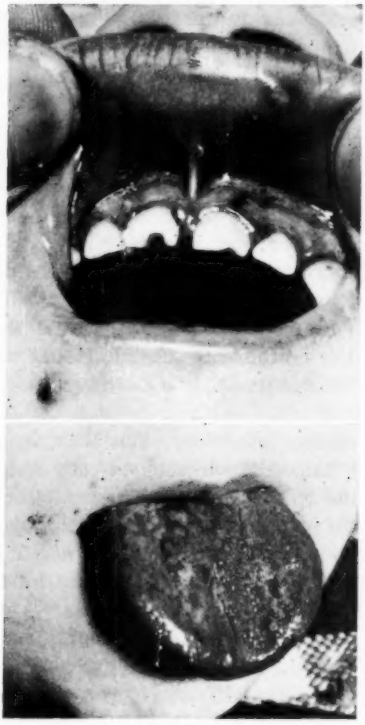

Fig. 10.

Fig. 9. Lesions of acute herpetic gingivostomatitis in a 2 year old child, demonstrating typical ulcers of the mucous membrane and swollen hyperemic gums. A cutaneous lesion is also present.

Fig. 10. Vesicles of acute herpetic gingivostomatitis involving the tongue of patient shown in Figure 9.

secondary infection the judicious use of one of the tetracyclines or penicillin in customary dosage may be useful against the bacterial invaders. Antibacterial therapy is not suggested for the case of average severity or in children who do not appear to have signs consistent with secondary infection, since antibiotics may be contraindicated on theoretical grounds.[11]

As for substances for topical application to the lesions, the possibility of traumatizing a tender and friable mucous membrane in an irritable and uncooperative child far outweighs any possible benefit derived from this approach. Therefore the use of astringents and gentian violet is not suggested. In the young infant the flow of saliva is usually sufficient to keep the lesions clean, and in the older child rinsing the mouth periodically with either cold water or normal saline solution may help in the removal of exudate from the surface of the ulcers. The course is usually entirely uneventful and recurrence unusual, if indeed it occurs at all.

Cortisone and ACTH should not be used during the acute stages of the disease because of the possibility of extension of the lesions, suggested by data concerning herpes infections in the experimental animal[9] as well as by data regarding cortisone in other primary virus infections.

Prophylaxis. In view of the high incidence of infection with this agent among young children, and the usual inapparent or extremely benign nature of the infection, the problem of prophylaxis among household contacts is relatively unimportant. Most of the contacts have probably encountered the virus previously and have established their own particular host-parasite relationship. Of the remainder, most of these will probably be infected by the time the case becomes clinically apparent. Other than reassurance to the mother that secondary cases among the family members are unlikely, although by no means impossible, and the use of a separate drinking glass, eating utensils and towel by the patient to reduce the size of the infectious dose delivered to other family members, attempts at prophylaxis are not indicated. It may be advisable, however, for the person with herpes simplex ("fever blisters") lesions to avoid close contact with infants and young children.

Differential Diagnosis. Some persons may have recurrent episodes of stomatitis indistinguishable from herpes on clinical grounds. Studies by Dodd and Ruchman[5] suggest that herpes simplex virus may not be the etiologic agent in these recurrent cases. The symptomatic therapy outlined is the same for both conditions, however.

The stomatitis described by Vincent[13] is often regarded as a synonym for herpetic stomatitis in the pediatric age group. With better methods for the identification of various infectious agents and further knowledge of the pathogenesis of many pharyngeal lesions it seems likely that the entity described by Vincent may have been due to a number of different agents. Certainly the clinical picture originally described is most unusual among children in the absence of some other well established etiologic agent.

Herpangina (Summer Sore Throat, Herpetic Sore Throat, or Vesicular Pharnyngitis)

This is a well defined clinical entity first described in detail by Zahorsky in 1920.[15] This report concerned a rather large series of cases observed during several summers in St. Louis. Although the lesions may resemble herpes simplex ulcers in some respects, thus suggesting the descriptive name, the herpes simplex virus is apparently not the causative agent. The recent studies of Huebner, Cole, Parrott and others[8, 10] have clearly shown that strains of Coxsackie virus immunologically related and defined as group A Coxsackie virus are responsible for this clinical disease.

Epidemiology. This infection is one of the large group in which the epidemiological behavior of the organism is nearly as characteristic as the lesions produced by it, and both aid the physician in establishing the clinical diagnosis with some degree of certainty. The age groups characteristically involved are infants and young children, with relatively few cases among older children and adults. This age pattern of the clinical disease, together with its epidemic appearance at times, suggests that the Coxsackie viruses responsible are probably highly infectious and may spread readily through the population. It also seems likely that those agents often occur, if not in the form of an entirely inapparent infection, as an extremely mild or at times atypical illness. Outbreaks of this disease customarily have been limited to the summer and early fall.

Clinical Manifestations. The initial complaint is usually fever, which may be variable, ranging as high as 103° F. or higher. After the appearance of the fever the older child often complains of sore throat, while anorexia or dysphagia may be noted in the younger patients. Less frequently, vomiting, abdominal discomfort and headache may also be noted. Subjective symptoms usually persist for three to five days.

Objective physical findings are limited to the mouth, where from two to 10 small grayish-white papulovesicular lesions may be noted. The size ranges from 1 to 4 mm., and the location most commonly involved is the anterior pillars of the tonsil, although other frequent sites are the soft palate and uvula.

The lesions themselves are surrounded by a red areola, and progress through the vesicular stage to ulceration during the course of three or four days. Healing is spontaneous and rapid, with complete recovery the rule by the end of one week.

Diagnosis. The diagnosis is usually evident clinically, since the presence and location of the vesicles and ulcers exclude the usual types of viral and bacterial pharyngitis. The limitation of the lesions to the posterior pharynx, their small size, the benign course and the seasonal

and epidemic pattern serve to distinguish this entity from herpetic gingivostomatitis.

The clinical laboratory is of little assistance in confirming the diagnosis. The isolation and identification of the specific virus involved is not necessary for accurate diagnosis of individual cases, however, since the clinical appearance is so characteristic.

Treatment. Therapy is limited to rest and symptomatic measures. The use of antibiotics presently available is not justified, since they have no effect on the etiologic virus. The occurrence of secondary bacterial infection does not present a problem in this disease. Indeed, the unfavorable alteration of the normal bacterial flora by these medications probably more than counteracts any possible rationale for their use.

Preventive measures are unknown, and are probably of relatively little importance because of the extremely mild nature of this disease under natural conditions.

Thrush*

This disease entity has been recognized for many years, and the causative agent, *Candida* (*Monilia*) *albicans*, has been studied extensively. Although this fungus is widespread in nature, it does not appear to be an important cause of disease except under certain circumstances. Those conditions favoring successful implantation with production of disease in the mouth, the usual site in infants and young children, are, first, age and general condition and, second, the use of antibiotics. The very young infant, particularly if debilitated by other pathological processes, is likely to be involved, as is the infant or child on prolonged "broad-spectrum" antibiotic therapy.

Clinical Manifestations. In the mild infection there are few, if any, complaints directly referable to thrush. The history often obtained is that of a young infant whose mother notes white flecks on casual inspection. Only when these lesions persist over the course of a day or more and resist gentle attempts at removal does the mother become concerned. At times the infant may appear somewhat irritable or may eat poorly, but in thrush of mild to moderate extent there is often no objective evidence of illness other than the presence of the oral manifestations. In cases with more severe involvement the infant may be acutely ill. This is particularly true of pneumonic or even more general involvement.

On physical examination the lesions appear as clear white plaques on the tongue, buccal mucosa, gums, palate or tonsil. The extent of the lesions may vary considerably, from a few small scattered patches to a

* Thrush in the newborn infant is also discussed on page 851.

nearly confluent plaque involving much of the interior of the mouth. The individual lesions may be wiped off only by causing considerable trauma to the underlying mucosa.

The natural history of the disease is that of a usually asymptomatic infection with lesions that may persist for a few days to two weeks or even longer in the more severe infection. In some patients, particularly in debilitated infants or in those receiving antibiotic therapy, the lesions may be more extensive and not subject to spontaneous improvement. Removal of the inciting cause together with vigorous therapy directed at the fungus is indicated in these cases.

Diagnosis. The diagnosis is based on the clinical recognition of the characteristic white patches. The agent may be easily identified by obtaining a small amount of the material from the lesions on a slide, adding a drop of 10 per cent potassium hydroxide and then examining it under the microscope. Confirmation of the diagnosis may also be made by culture of the material from the lesions.

Treatment. The classic therapeutic approach has been the topical application of a 1 per cent aqueous solution of gentian violet to the lesions. This is applied with cotton applicators using a rolling motion, and applications are made at 12 hour intervals initially, and once daily after improvement is noted. Recent therapeutic trials suggest that a 1:1000 aqueous solution of Zephiran gently applied at similar intervals using the same method is equally effective. The latter therapy is nontoxic and nonirritating, and staining of clothing and blankets does not result. Staining may be troublesome with gentian violet and represents the sole disadvantage of that medication. The effectiveness of Zephiran precludes the use of more irritating substances such as silver nitrate.

In the rare infant showing more extensive involvement, particularly with invasion of the trachea or lung, systemic administration of Mycostatin is indicated.[1]

Stomatitis and Diarrhea of Infants

This clinical disease process was first recognized by Buddingh and Dodd[3] in 1942. Although on clinical grounds it appeared to resemble herpes gingivostomatitis, a filterable virus was isolated which did not appear to be herpes simplex virus. This agent was transmitted successfully to the cornea of young rabbits, but has subsequently been lost, and no other isolations of a similar agent are known.*

Although the majority of the cases described were in young infants, occasional cases were recognized in older children. Adults were found to harbor the infection, usually without symptoms.

* Katherine Dodd: personal communication.

The *symptomatology* was limited to refusal of feedings and sore mouth, with little or no fever. In many of the infants a moderate diarrhea was noted. The physical findings included numerous vesicles involving the tongue, gums and the inner surface of the lower lip.

The *course* of the disease in the cases described was uneventful, with recovery in three to 10 days. No effective therapy or prophylactic measures are known.

The frequency of this type of infection is unknown. The clinical similarity to the usual herpetic gingivostomatitis is striking. At this time there is no method of distinguishing between these entities by techniques available in the clinical laboratory. Consequently it may be suspected that this type of infection is probably often diagnosed as herpetic in origin. However, since therapy is identical for either, the separation of these entities appears to be chiefly of academic interest.

ORAL MANIFESTATIONS OF INFECTIOUS DISEASES NOT PRIMARILY INVOLVING THE MOUTH

Many of the systemic communicable diseases encountered in the pediatric age group have lesions which are often characteristic and sometimes even diagnostic of that particular disease. These oral lesions often aid the physician in the recognition of the disease, and thus assist in planning therapy. Although other examples are available, the best example of this group is that of measles.

Measles

The oral lesion (enanthem) identified with rubeola is the Koplik spot, which has been recognized for at least 60 years. This lesion is perhaps one of the most characteristic of all the individual physical signs of disease processes, and it is most unusual for the experienced physician to be misled by its appearance. The lesion itself is a small white papule approximately 1 mm. in diameter, surrounded by a red areola. The most characteristic site is on the buccal mucosa, opposite the molar teeth, although there may be considerable variation in the location. At times only a few lesions are noted, while at the other extreme the buccal mucosa may be literally covered with lesions and may appear as though it had been heavily salted. The usual time of appearance of the Koplik's spots is from about 11 to 13 days after effective exposure. They usually precede the rash from a few hours to a day or two, and rarely persist longer than a day.

A second type of enanthem may also be seen in measles. This consists of a red macular rash involving the soft palate, and occurs at the time of onset of the exanthem. This is a curiosity only, since the rash,

catarrhal signs, fever and the Koplik's spots are much more important in reaching a diagnosis.

Diagnosis. The diagnosis is usually apparent from the appearance of the lesions, although the presence of the fever, cough and coryza of the measles prodrome are reassuring when making an early diagnosis of measles. The measles rash is of little diagnostic help at this stage of the disease, since the Koplik's spots are usually fading by the time the rash appears.

Treatment. No therapy is suggested at this stage, since gamma globulin has not been demonstrated to be of value in the modification of the disease or its complications after symptoms are present. The use of antibiotics does not look particularly encouraging in the prophylaxis of secondary bacterial infection,[14] although further studies are needed. This should not be interpreted to indicate that antibiotics should not be used in the therapy of secondary bacterial complications, since these drugs may be lifesaving in this situation.

Varicella

An often overlooked site of the vesicles of chickenpox is the mucous membrane of the mouth. The oral lesions may be absent in the mild case, but may be extensive and cause considerable discomfort in the child with more severe varicella. The appearance of the oral lesions is similar to that of the vesicles on the skin, with one exception. The oral vesicular lesions progress somewhat more rapidly to form small round ulcers instead of the gradual transition to the crust stage of varicella as seen on the remainder of the body. Discomfort, if noted, is usually limited to the first three or four days of the disease.

Diagnosis of the cause of the oral lesions is dependent on the presence of the characteristic cutaneous manifestations of varicella, and no technique currently available in the clinical laboratory is of assistance in identifying this type of oral lesion.

Treatment of the oral lesions, as of the uncomplicated case of varicella, is entirely symptomatic.

Scarlet Fever and Streptococcal Pharyngitis

As might be expected from the dual pathogenesis of the various symptoms and signs of this disease complex, the oral lesions present more than a single manifestation. The more common oral manifestation in both scarlet fever and streptococcal pharyngitis is the presence of an exudative pharyngitis. The quality present may vary from a scanty, thin exudate, barely recognizable as such, to a thick, dull white pseudomembrane clinically indistinguishable with certainty on the basis of

appearance alone from diphtheria and infectious mononucleosis. This exudative reaction is most often noted on the tonsil, but the lymphoid tags after tonsillectomy may be involved as well as the posterior pharynx.

The second type of oral manifestation is due to the local absorption of erythrogenic toxin produced by the responsible streptococcus during the early course of scarlet fever. This includes a macular rash usually involving the palate and is more commonly noted in scarlet fever. This rash may appear somewhat hemorrhagic or petechial at times. An additional manifestation of the toxin absorption is the typical strawberry tongue of scarlet fever, with hypertrophy of the papillae and subsequent desquamation.

The *diagnosis* is dependent on the remainder of the clinical picture together with the isolation of group A beta hemolytic streptococci from the pharynx. With the presence of the typical rash, often accompanied by a chill and vomiting, the diagnosis is evident. In addition to the presence of exudate, other symptoms such as fever, chill, cervical adenitis, absence of coryzal symptoms, severe sore throat and abrupt onset have been found helpful in the clinical recognition of streptococcal pharyngitis in the older person. It has been our experience that these symptoms and signs tend to be less helpful in the infant and young child, however.

Therapy consists of a full 10 day course of penicillin, achieved by any one of several methods. Perhaps the most convenient technique at present is the use of oral penicillin G in doses of 750,000 to 1,000,000 units a day given in either three or four divided doses, avoiding administration at mealtime. If pencillin V is used, the latter precaution is unnecessary, and perhaps slightly lower doses may be possible. Repeated daily injections of 300,000 units of procaine penicillin (or every third day for three doses if an aluminum monostearate in oil preparation is used) or a single injection of 600,000 units of benzathine penicillin G represent acceptable alternatives to the oral medication.[16]

Infectious Mononucleosis

The reason for including this disease with the others in this category is the problem of recognition of the so-called anginose type of involvement.

In some patients with infectious mononucleosis the presenting complaints may be fever, severely sore throat and malaise. Initially the physical findings in this type of involvement may be limited to the pharynx, with the presence of a thick, dull white pseudomembrane covering the tonsils and at times involving the lymphoid tissue of the posterior pharynx as well. This membrane (Fig. 11) is limited to

the lymphoid tissue and does not extend to the surface of the palate or other oral mucosa. The presence of generalized lymphadenopathy and an enlarged spleen are helpful clinical findings if present. However, the cervical adenitis of "bull neck" diphtheria may closely resemble the severe cervical adenitis occasionally seen in infectious mononucleosis.

The specific *diagnosis* of this disease is dependent on the clinical laboratory, and is based on the presence of the atypical mononuclear cells in the peripheral blood and an increase in the heterophile antibody titer during the course of the disease. It should be noted that the lymphocytosis characteristic of infectious mononucleosis may be absent during this early stage of the disease. Careful and often repeated cultures are necessary to rule out the presence of diphtheria and streptococcal pharyngitis with certainty.

Fig. 11. Dull white pseudomembrane of infectious mononucleosis present on upper pole of left tonsil.

Therapy is entirely symptomatic, and is limited to bed rest and the use of aspirin or codeine and a soft diet for the pharyngeal discomfort, if intense.

Diphtheria

Although this disease is most unusual among adequately immunized children, and its incidence is extremely low in this country, it still occurs with sufficient frequency to demand serious consideration in the differential diagnosis of any child exhibiting a pseudomembranous type of pharyngitis. Diphtheria is excluded only by adequate cultures from beneath the margin of the lesion utilizing Löffler's or similar media

together with careful clinical evaluation for signs of toxin absorption. Since the complexities of the proper diagnosis and treatment of this disease are beyond the scope of this presentation, the reader is referred to other sources.

Bednar's Aphthae

The traumatic lesions which are occasionally noted on the hard palate and are often referred to as Bednar's aphthae sometimes cause confusion with oral infections. These characteristically occur as one or more small, superficial ulcerative lesions on the hard palate of the young infant, and appear to be related to nipple trauma or other similar sources of irritation. No therapy is required, and the ulcers usually heal promptly after removal of the source of irritation.

SUMMARY

A brief review of some of the more commonly encountered oral manifestations of infectious diseases has been presented. An attempt has been made to describe in some detail some of the more common infectious diseases either limited to the mouth or with prominent clinical manifestations in this area. Some of the points in the differential diagnosis and suggestions for current therapy for this group of infections have been included.

The photographs were prepared by the Department of Photography, State University of New York Upstate Medical Center.

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State University of New York
Syracuse 10, New York

ORAL NEOPLASMS OF CHILDREN

HAMILTON B. G. ROBINSON, D.D.S.

The oral region of children is the site of neoplastic growths of benign and malignant types. In addition the leukemias may show oral manifestations, particularly enlargement of the gingivae and necrosis of the soft tissues, as relatively early signs.[26] Since the leukemias are not tumors of the oral tissues, but simply present oral symptoms, they will not be discussed here, but they should not be overlooked. The oral regions may be involved by numerous types of benign neoplasms and by malignancies. The prognosis of malignant neoplasms usually is grave in children,[9] and because of the frequency of lymphadenopathy associated with infectious disease in children, symptoms may be ignored.[16, 28] For simplicity in discussion these different neoplasms will be divided into benign and malignant tumors of the soft tissues and of bones. It is recognized that this discussion is not exhaustive, but the relatively less rare neoplasms are discussed and their non-neoplastic counterparts presented where diagnosis is a problem.

BENIGN TUMORS OF THE SOFT TISSUES

The soft parts of the mouth, the lips, cheeks, floor of the mouth, tongue, gingiva and lining membranes of the palate may be the sites of origin of many benign tumors.

Papilloma

The papilloma (Fig. 12) may occur on any of the covering epithelia of the mouth, and usually presents a pedunculated verrucous appearance varying from a smooth surface to a more characteristic velvety or

cauliflower appearance. The surface ordinarily is hyperkeratinized, resulting in a white color. Diagnostically, the most frequent lesion of similar appearance is *verruca vulgaris* (Fig. 13). These warts are probably of virus origin, appear to be transmissible, and may respond to conservative therapy (application of 20 to 30 per cent hydrogen peroxide, daily, for several days). Their most frequent site is the lip, although they may also be found on the tongue and other oral tissues.

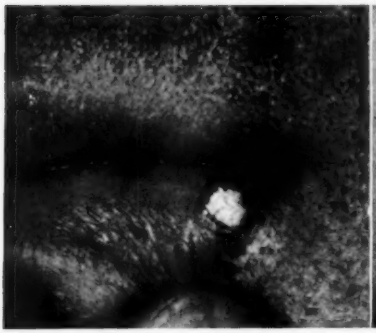

Fig. 12.

Fig. 13.

Fig. 12. Papilloma of the lower lip. The lesion is white with a verrucous surface. (From Thoma and Robinson.[35])

Fig. 13. Verruca vulgaris of the upper lip. The lesion is white with a somewhat verrucous surface, but, as in this case, may appear less "warty" than a papilloma. (From Thoma and Robinson.[35])

Fibroma

The fibroma may be found in any of the oral regions (Fig. 14). It may be sessile or pedunculated, usually with a smooth surface and varying in color from that of the surrounding normal mucosa to a pale pink. The surface may become keratotic as the result of chronic irritation from food and other foreign material in the mouth rubbing the elevated surface. Microscopically, the lesion may show a predominance of young fibroblasts or collagenous fibers. There may be various admixtures of other types of mesenchymal tissue as bone (osteofibroma), blood vessels (hemangiofibroma) (Fig. 15) or fat (lipofibroma). When the tumor shows a loose connective tissue arrangement, the term "myxofibroma" is applied. The term "peripheral odontogenic fibroma"[2] has been used to designate a fibroma originating "within the periodontium." It is admitted that it is only academic to labor the point of origin, and therefore they will not be discussed.

All the oral fibromas should be removed surgically, extending the

excision to include the base. When the lesion is on the gingiva, among some the removal of the adjacent teeth is routine, but that procedure is now more generally considered optional. In most instances the fibroma is widely excised and the patient observed for possible recurrence indicating incomplete removal.

Fig. 14. Fibroma of the gingiva. Clinically, this is an epulis. It is soft, freely movable, red with a few yellow patches (due to ulceration and secondary infection) and not tender.

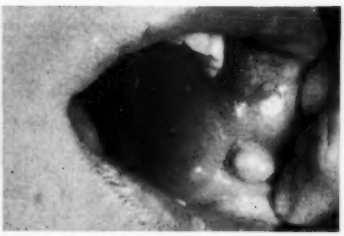

Fig. 15. Fig. 16.

Fig. 15. Hemangiofibroma of labial mucosa. The lesion was a deeper red than the surrounding mucosa. (From Thoma and Robinson.[35])

Fig. 16. Irritation hyperplasia (irritation fibroma) on the buccal mucosa of a chronic cheek chewer.

Irritation hyperplasia[7, 35] (irritation fibroma) may appear clinically as a fibroma. These lesions arise as a result of irritation, from cheek chewing (Fig. 16), tongue chewing or trauma from food. Their most frequent site is the cheek. The treatment is the same as for fibromas with the added necessity of eliminating or minimizing the trauma.

The term "epulis" is applied to benign neoplasms and hyperplasias of the gingiva. It is not a specific term, and the differentiation into fibromas, irritation hyperplasia, giant cell lesion, and others, is based on more precise diagnostic techniques, including biopsy.

More generalized enlargements of the gingivae include leukemic reactions,[7, 26, 35, 37] Dilantin hyperplasia[32] (Fig. 17), hormonal enlargements of puberty[10] and hereditary gingival fibromatosis.[7, 17]

Pyogenic Granuloma

Pyogenic granuloma is not a neoplasm, but may readily be mistaken for a neoplastic growth. It occurs as an elevated lesion, sessile or pedunculated, usually soft and redder than the normal mucosa. They frequently are ulcerated and bleed readily. The gingiva is the most

Fig. 17. Dilantin hyperplasia. The gingivae commonly become greatly enlarged in epileptic patients receiving therapy with Dilantin Sodium.

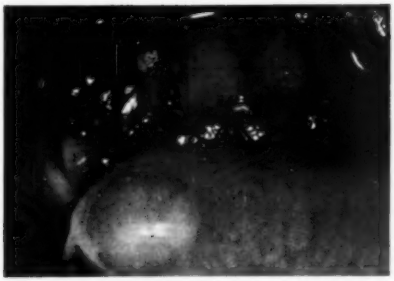

Fig. 18. Peripheral giant cell reparative granuloma on maxillary right gingiva of 7 year old boy. Clinically, this is an epulis.

common site, but they also appear on the lips, tongue, buccal mucosa and in extraction sockets, usually following minor trauma.[20]

Reparative Giant Cell Granuloma

Reparative giant cell granuloma[3] (Fig. 18) long was known as the giant cell tumor or giant cell epulis and earlier as the giant cell sarcoma. They are benign lesions occurring on the gingiva and probably associated with trauma and exaggerated repair rather than with neoplasia. Although efforts have been made to associate these lesions with the abundant giant cells present during the resorption of deciduous teeth,[13] more careful analysis of cases has shown that the tumors may

occur in the molar regions where no deciduous teeth were present and that the average age at first examination (32.1 years) is older than formerly believed.[2]

Neurogenic Fibroma

Neurogenic fibroma may occur in the oral regions of children, especially on the tongue, although the palate, cheeks, lips and gingivae are also affected.[2] They simply appear as nodules or swellings that may reach large size.[7] Multiple nodules may occur in von Recklinghausen's disease.

Schwannoma

Schwannoma may appear in the soft tissues of the oral cavity and on the lips. It is benign and produces a painless swelling, but may lead to facial palsy.[34] Local incision is indicated.

Nevi

Nevi, or moles, are benign lesions which may be cellular or vascular and pigmented or nonpigmented. Both types are rare on the mucous membranes. A nonpigmented nevus of the gingiva[11] has been reported, but its repetitive use[2, 35] to illustrate the lesion is indicative of its rarity.

Myoma

Myomas of either smooth or striated muscle types are extremely rare in the oral cavity and have usually been reported in adults when discovered.

Myoblastic myoma (myoblastoma, granular cell tumor) is relatively frequent in its oral occurrence. One type, known as congenital epulis, is present at birth and attached to the alveolar mucous membrane by a pedicle.[8] It is easily removed and does not recur. Because of the appearance of epithelium in some of these lesions they have been suggested as being odontogenic in origin. Since no odontogenic tendencies or potentialities have been demonstrated in these lesions, as in ameloblastoma, the possibility of an odontogenic origin is remote. Myoblastic myoma is also observed as a fairly firm mass in the tongue, often with a keratinized superficial epithelial covering, but at an average age of 40 years.[2] The question of whether they are neoplastic or not has not been settled.[19]

Lipoma

Lipoma may appear on the tongue, cheek, alveolar gingiva, floor of the mouth or in the salivary gland. It has even been reported congenitally in the cheek. Typically it is soft, yellow and freely movable. Blood vessels may be seen traversing its yellow surface. It is benign, but may grow to considerable size.

Hemangioma

Hemangiomas may be found in any of the soft tissues of the mouth. The lip is a frequent site for either capillary or cavernous types. They also are found on the tongue, the floor of the mouth, labial and buccal mucosa and on the gingiva (Fig. 19). The lesion is usually dark blue

Fig. 19. Hemangioma of gingiva. The maxillary right side shows a deep red discoloration along the gingival margin. In the molar region this port-wine red lesion flared out over the entire gingiva.

or black, soft, and the blood is readily evacuated by stroking the surface. They may vary from small, localized lesions to large lesions covering large areas of the oral cavity. In Sturge-Weber syndrome a hemangioma may involve one side of the face with jacksonian seizures on the opposite side because of vascular disturbance of the pia and cerebrum on the side of the hemangioma.

Lymphangioma

Lymphangioma may involve the tongue (Fig. 20), cheek or other oral tissues. The entire tongue may be involved, or the lesion may be limited. The appearance is usually that of small bunches of grapes, producing a nodular yellow to grayish-pink surface. Both hemangiomas and lymphangiomas may be excised if circumscribed, but the larger lesions are more difficult to handle. Sclerosing agents[4] may be injected or radiation may be used, although response to the latter is variable.

Hygroma colli congenitum refers to a cystic lesion of the neck,

present at birth, but often remaining small for several months before it becomes large and distended.[7]

Hemangio-endothelioma

Hemangio-endotheliomas occur as benign tumors in children (infantile benign hemangio-endothelioma).[33] They appear as soft, irregular masses in the lip, tongue or other oral tissues. Some have considered them potentially malignant.[6]

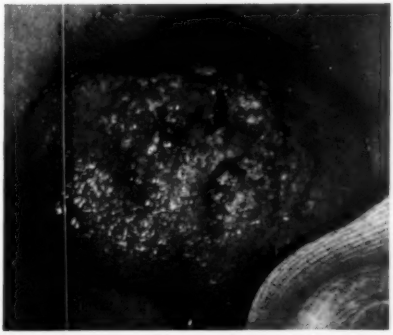

Fig. 20. Lymphangioma of tongue. This extensive lesion has a yellow cast and is elevated and soft.

MALIGNANT TUMORS OF THE SOFT TISSUES

Squamous Cell Carcinoma

Squamous cell carcinoma of the soft tissues of the mouth is relatively rare in children. However, any wartlike lesion, ulcer, fissure, crusting, hyperkeratosis or swelling which is persistent should be thoroughly investigated. Whenever a definite diagnosis cannot be established from clinical examination and history, biopsy is indicated. It has been pointed out that carcinoma in young children does not follow the distribution of areas of chronic irritation (lower lip, anterior two thirds of tongue, buccal mucosa) that it does in adults, but is more indiscriminately located.[16]

Transitional Cell Carcinoma

Transitional cell carcinoma (lympho-epithelioma) may arise in the nasopharynx, the base of the tongue, palate or the maxillary sinus and involve the oral cavity. It may be found in relatively young persons. Ulceration of the palate may be the earliest symptom, or, when located in the maxillary sinus, osteolytic activity due to pressure may give a

resemblance to a dentigerous cyst, radiographically. The first symptom, however, may be lymphadenopathy with nasal obstruction and possibly ear ache. Although the tumor is quite radiosensitive, its tendency to wide metastasis makes the prognosis extremely grave.[34]

Fibrosarcoma

Fibrosarcoma, though rare in the oral region, is found most commonly in children when it does occur. If it forms at the chin or angle

Fig. 21. Lymphosarcoma of jaw of 3 year old girl. The untreated lesion has destroyed and expanded large areas of bone.

of the jaw, it results in deformity of the mandibular contour. It may produce an osteolytic defect by invasion. Intraoral fibrosarcomas may appear as swellings with early ulceration on the gingiva, hard palate or tongue. Radical resection is indicated, but even then the prognosis is poor.[34]

Lymphosarcoma

Lymphosarcoma may involve the oral cavity and most frequently arises in the region of Waldeyer ring. It is one of the malignant lymphomas, and some authors tend to classify reticulum cell sarcoma (see p. 901) and Hodgkin's disease with it. In the oral cavity lymphosarcoma may be primary, 20 such cases being reported.[2] It may first resemble nonspecific oral inflammation, but may grow rapidly to reach considerable size[4] (Fig. 21).

Hemangio-endothelioma

Hemangio-endothelioma may be a malignant neoplasm in a child. The lesion may appear quite benign and metastasize later, but such results are extremely rare. A case reported in a seven weeks old infant resulted in death at 15 weeks.[5]

Melanoma

Fortunately, melanoma is rare in the oral cavity, only 61 cases of primary oral origin being reported.[15] It should be recalled that non-pathologic pigmentation of the oral mucosa is usually seen in dark races and is not rare in white races.[35] Jehger's syndrome (intestinal polyposis with peroral pigmentation) may also result in intraoral discoloration.

BENIGN TUMORS ON THE JAW BONE

Osteoma

Osteomas may occur in the maxilla or mandible. The oral cavity is the site of certain bony hyperplasias that occur so frequently as to be

Fig. 22.

Fig. 23.

Fig. 22. Torus palatinus. This hard, not tender elevation is found in the midline of the palate.

Fig. 23. Torus mandibularis. The enlargements seen bilaterally on the lingual aspect of the mandible are hard and radiographically are opaque.

classified as "normal." The torus palatinus (Fig. 22) is found as a radiopaque, bony protuberance in the midline of the palate of one out of five adults. It begins to grow early in life, and both the incidence and size increase during the first three decades. In 13.7 per cent of children under 10 the lesion is observed, in 19.5 per cent of those between 11 and 20 years of age, and 23.9 per cent in the third decade.[21] Similar bony enlargements are found on the lingual aspect of the mandible in the premolar region (Fig. 23). They usually are bilateral

and may be lobulated. Only 1.5 per cent of children under 10 years old showed this lesion, while in the next decade they rose to a 4.9 per cent incidence, and a 10.5 per cent incidence in the 21 to 30 year group.[21] Osteomas are much rarer than these osseous enlargements, but histologically and clinically they are similar in appearance, being differentiated by site.[27] This admittedly is an arbitrary differentiation.

Chondroma

Chondroma is an infrequent finding in the jaw bones, but may occur within the bone or on its surface, more often on the mandible than on the maxilla. It may be found at the condyle or the mandibular symphysis. It usually is radiolucent in comparison with the adjacent bone. Radical excision is indicated because of possible relationship to chondrosarcoma.

Central Fibroma

Central fibroma may appear as a radiolucent area within the bone. Some of these are considered to be odontogenic in origin (especially if epithelium-resembling developing dental structures are present). Central fibromas are usually asymptomatic and when discovered in routine dental radiographs may be mistaken for cysts (see p. 897). They are treated by simple surgical excision.

Central Giant Cell Reparative Granuloma; Giant Cell Tumor

Central giant cell reparative granuloma usually is discovered in the radiograph as a radiolucent area, sometimes with thin radiopaque septa. However, swelling may be the first symptom. It is now assumed that trauma is often associated with its origin. These lesions are found primarily in young persons; Waldron noted that two of 28 cases of "giant cell lesions" of the jaws were in the 0 to nine year age group and 13 additional in the 10 to 19 year age group.[36] We have considered these lesions one group in the series of reactions to bone injury observed in the jaws (Fig. 24).[27] Histologically, giant cells are observed in a loose, edematous stroma. Often small foci of osteoid or bone are seen.

True giant cell tumor of bone is much rarer in the jaw bones than are the central giant cell reparative granulomas. Waldron, in his series of cases, considered five to appear identical, histologically, with giant cell tumor of bone. The ages of these patients were seven, 10, 10, 10 and 11. This is distinctly unlike the age distribution in giant cell tumor found in other bones. No case of malignant giant cell tumor of the jaws has been recorded in the literature.[36] It is probably safe to assume

that if true giant cell tumors do occur in the jaws, they are extremely rare or are not biologically similar to those in other bones. Thorough curettage appears to be adequate treatment for the giant cell lesions, for the prognosis is good.

Fibrous Dysplasia

Fibrous dysplasia has been described in the jaws by Schlumberger[29] and others. The question of whether this lesion represents a true fibrous dysplasia related to that described by Lichtenstein,[22] Jaffe,[18] and Albright[1] or is a form of ossifying fibromas has been debated frequently.[27] From the clinical viewpoint this is somewhat academic. Lesions occur in the oral region, characterized by slow, insidious, firm expansion with symptoms of numbness appearing occasionally. The

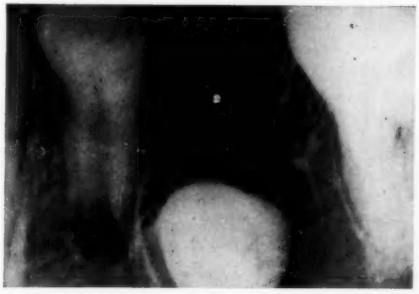

Fig. 24. Giant cell reparative lesion associated with an erupted tooth and probably preventing its eruption. (From Robinson.[27])

maxilla is more commonly involved, although the lesion may appear in the mandible alone, or in both the mandible and maxilla. The malar bone may also be involved. This slowly expanding swelling usually begins before puberty and results in facial deformity. The radiographic appearance varies, always showing expansion of bone, but with a diffuse radiolucency, mottling, cystlike areas, or a "ground-glass" appearance. Histologically, the bone marrow is replaced by fibrous tissue.

An unusual complete facial involvement has been reported under the name of "cherubism." In four or five children in one family the lesions appeared multilocular, radiographically, and the distortion of the face gave them a peculiar "cherubic" appearance.[34]

Robinson[27] has regarded many of these jaw lesions called fibrous dysplasia as reaction of the bone to injury. The jaws are peculiar, in that they contain 20 deciduous teeth or 32 permanent teeth, each capable of individual trauma and infection. Disturbed reaction of bone (dysplasia) during childhood and after injury may lead to expansion of the bone. Many cases of this type probably have been classified as

fibrous dysplasia, although unrelated to the generalized bone disease given this name. We prefer to call this disturbed reaction to injury "*osseous dysplasia*" to differentiate it from the fibrous dysplasias associated with faulty embryogenesis.

Thoma[34] classifies *fibro-osteomas* as true neoplasms starting by proliferation of fibroblasts in the bone marrow and differentiates them, clinically, by their sharply circumscribed appearance. When the tumor is predominantly fibroblastic, he uses the term "osteo-fibroma"; when there is considerable osteoid or bone, he uses the term "fibro-osteoma." These neoplasms occur with a somewhat older age group than the fibrous dysplasia or osseous dysplasias, although any of these dysplasias and neoplasms may occur at widely different ages. The term "ossifying fibroma" has also been applied to these lesions, some of these probably representing those now classified as fibrous dysplasia, other osseous dysplasia and still others fibro-osteomas.

The localized lesions, if circumscribed, are removed surgically and show little tendency to recurrence. The deformity usually regresses or disappears after removal of the central lesion. The generalized lesions are treated cosmetically. Radiation is of questionable value. If local infection exists, it should be eradicated. The osteogenic tissue seems to mature or to reach a state of equilibrium beyond which further growth is not apparent. None of these osseous lesions become malignant.

Eosinophilic Granuloma

Eosinophilic granuloma often involves the bones of the jaws. It is not considered a neoplasm, in the strict sense, but clinically it reacts as a neoplasm. In general, opinion places it as an inflammatory reaction of bizarre type or as a variant of Schüller-Christian disease. The lesions, more common in the mandible than the maxilla, involve the bone in a destructive process resembling the osteolytic activity of osteomyelitis or of a giant cell reparative granuloma (Fig. 25). It is found primarily in children and young persons. The lesion responds to x-ray therapy.

Ameloblastoma

Ameloblastoma (adamantinoma, adamantinoblastoma) is an epithelial tumor arising from the odontogenic apparatus of the cells with a potentiality for forming tissues of the enamel organ. Its structure resembles histologically that seen in certain developmental stages of the enamel organ.[24] The average age of onset (discovery) of the tumor is 30 to 32 years, but 24 were discovered in the first 10 years of life and 113 in the next 10 years in 1036 cases surveyed from the literature.[31]

The appearance of the lesion varies, clinically, radiographically and histologically. Histologically, the tumor may be solid, showing a struc-

ture of odontogenic-type epithelium in a cellular connective tissue stroma (Fig. 26), but may change to a multicystic lesion with relatively little fibrous stroma.[23] Clinically, the first sign may be a swelling of the face (Fig. 27). However, many ameloblastomas are first discov-

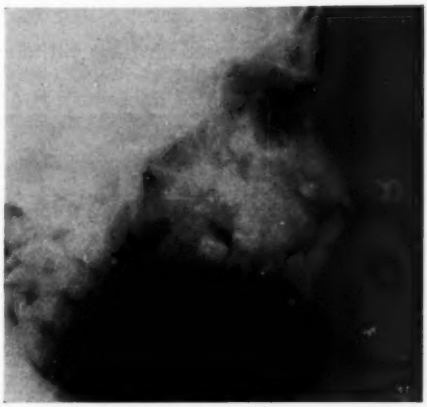

Fig. 25. Eosinophilic granuloma producing marked generalized radiolucency in the mandible. The inferior border appears only as a fine radiopaque line.

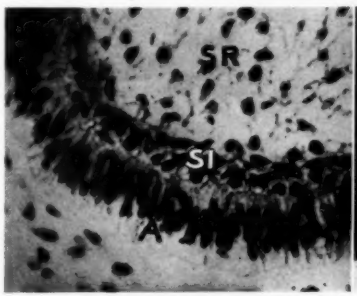

Fig. 26.

Fig. 27.

Fig. 26. Ameloblastoma of mandible. This microscopic section, from the lesion seen in Figure 27, shows the cells suggestive of the enamel-forming apparatus. *A*, Ameloblast-like cells; *SI*, stratum intermedium-like cells; *SR*, stratum reticulum-like cells.

Fig. 27. Ameloblastoma of mandible of a 2½ year old child. The swelling of the jaw is suggestive of an infection of the salivary glands or jaw bone.

ered in routine radiographs or because of radiographs taken for localized enlargements of the alveolar regions (Fig. 28). Ameloblastomas are found four times as frequently in the mandible as in the maxilla. The radiographic appearance is inconstant. Whenever one sees a radiolucency of the jaw with lobulated borders (Fig. 29) or loculations (Fig. 28), ameloblastoma should be suspected. However, a final diag-

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nosis of ameloblastoma should not be made by radiographs alone, since dentigerous cysts (Fig. 30), traumatic bone lesions, central giant cell lesions and other lesions may produce radiolucencies resembling ameloblastomas. It has been suggested that many ameloblastomas may arise from dentigerous cysts. Although this may occur (Fig. 31), it apparently is rare.

Ameloblastomas should be removed in their entirety. They tend to

Fig. 28.

Fig. 28. Intraoral radiograph in maxillary premolar region showing radiopaque trabeculae forming multicystic-appearing radiolucencies, suggestive of ameloblastoma. This was confirmed by biopsy.

Fig. 29.

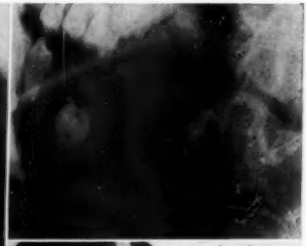

Fig. 30.

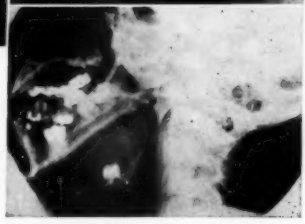

Fig. 29. Ameloblastoma of mandible of child. The lobulated, multiloculated radiolucent lesion has displaced the adjacent teeth.

Fig. 30. Dentigerous cyst of mandible in child. The molar tooth about which the cyst formed is depressed. Compare with Figure 29.

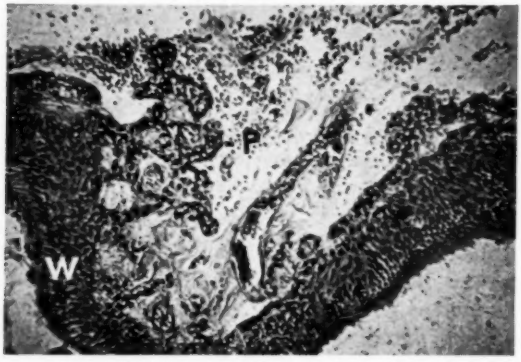

Fig. 31. Dentigerous cyst wall. The wall of this dentigerous cyst (W) shows proliferations (P) suggesting metaplasia to a neoplasm (ameloblastoma).

recur frequently, over one third with follow-up showing recurrence.[31] As many as 24 recurrences have been reported in a single case.[24] They are relatively radioresistant, although some success has been claimed. Although about 4.5 per cent have been reported as malignant (metastasis or metaplasia),[24] generally they should be considered persistent tumors rather than malignancies.[24]

Some effort has been made to subclassify ameloblastomas into primitive, plexiform, stellate, follicular, basal cell and acanthomatous types.[34] Personally, I feel that this is an academic exercise of little or no clinical importance. A *hemangio-ameloblastomatous* type has also been reported. The so-called *melanoameloblastoma* may represent a *retinal anlage tumor*.[30]

The *mixed odontogenic tumor*, or *odonto-ameloblastoma*, is differentiated because intercellular substance is deposited within the tumor. This calcified material is in the form of small teeth or tooth-material masses. The clinical and radiographic appearance (Fig. 32) is similar to the ameloblastoma, and their clinical course is the same.

Fig. 32.

Fig. 33.

Fig. 32. Mixed odontogenic tumor (odonto-ameloblastoma) of mandible showing trabeculated pattern similar to, or identical with, ameloblastoma. (From Thoma and Robinson.[35])

Fig. 33. Compound composite odontome in maxillary canine region. It is made up of several calcified dental masses. Intraoral radiograph.

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Odontogenic (ameloblastic) *fibromas, myxomas* and *neurinomas* are recognized by some oral pathologists,[2, 14] but their differentiation from similar nonodontogenic tumors in the same region is tenuous and of questionable clinical value.

The *odontomas* (Fig. 33) are not true tumors, but are benign, developmental anomalies of the teeth.

Myxoma

Myxomas are relatively infrequently observed, but may appear in the maxilla or mandible. In the jaws they usually follow a benign course, and metastases may occur. We recently observed a myxoma of the maxilla in a six year old girl. Clinically, it had distended the maxilla and the face, similar to a fibrous dysplasia or osseous dysplasia. Radiographically, radiopacity was seen with trabeculae of radiopaque material resembling ameloblastoma and osseous dysplasia. These are the typical objective symptoms of myxoma of the jaws. This tumor was well circumscribed and enucleated with relative ease. Because of their benign course in the jaws an odontogenic origin has been suggested,[2] but has not been substantiated.

MALIGNANT TUMORS IN THE JAW BONE

Osteogenic Sarcoma

Osteogenic sarcoma is used as a general term for malignant tumors whose ancestor cells are of a type that, when fully differentiated, would be osteoblasts. Osteosarcoma, chondrosarcoma, myxosarcoma and other subtypes are included, but, since many single tumor masses may show different cell patterns in different areas, the specialized classifications are not of great value. Osteosarcoma is rare, but is found in young children and is most common in the teen-age and subteen-age group.

Unlike most other malignancies, osteosarcoma may be painful in its early stages. This, coupled with radiolucency seen in some types of osteogenic sarcoma, may lead to a misdiagnosis of osteomyelitis. The radiographic picture varies in some stages. The bone may have a mottled appearance, in others a definite radiopacity with a "sun-ray" effect at its borders may be evident, while in periosteal types the "sun-ray" effect may be seen at the border of the bone, particularly the mandible. Expansion of the bone may not be an early sign.

Osteogenic sarcoma is more frequent in the mandible than in the maxilla. In any child or young patient with loosening of a permanent tooth, or teeth, or premature loosening of deciduous tooth, or teeth, intraoral x-ray examination is indicated. A "spiking" of the roots

(Figs. 35, 38) is strongly suggestive of malignancy, especially when accompanied by loss of the lamina dura of the alveolar socket, or mottling of the supporting bones.

Fig. 34. Fig. 35.

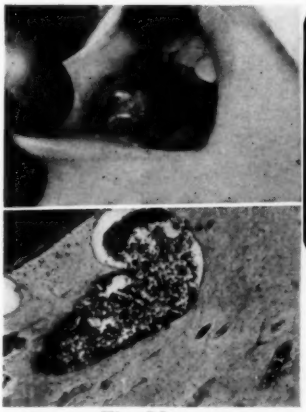

Fig. 34. Mandibular molar region of 12 year old boy with metastatic chondromyxosarcoma. The lesion was fairly soft and dark red, and there were areas of necrosis on its surface.

Fig. 36.

Fig. 35. Mandibular molar region of 12 year old boy with metastatic chondromyxosarcoma. The roots of the second molar are spiked, the distal root of the first molar is spiking, and the lamina dura is lost.

Fig. 36. Metastatic lesion of chondromyxosarcoma in blood vessel of pulp of second molar tooth seen in Figure 35.

Reticulum Cell Sarcoma

Reticulum cell sarcoma may be primary in the mouth, or metastatic lesions may involve the jaw bones (see Figs. 37, 38). Those primary in the jaw are rare, only eight being reported in the literature. One was in a nine year old boy and one in a 14 year old boy. They respond well to radiation therapy.[12]

Myeloma

Myelomas may be found centrally in the jaw bones. *Endothelial myeloma* (Ewing's tumor) occurs in early years and may be primary in the mandible, usually the ramus, or the maxilla, usually the sinus. It may produce a swelling of the face, with expansion of the cortical bone. Peripheral osteophytes may be observed radiographically. Ewing's tumor is relatively radiosensitive, but prognosis should be reserved. *Multiple myeloma* (plasma cell myeloma) is usually a tumor of later life.

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Metastatic Tumors

Carcinoma, neuroblastomas, sarcomas, sympathicoblastoma and hypernephromas are among the tumors that have been reported as metastasizing from bone, ovary, testes, breast, lung, kidney, adrenal, rectum and thyroid to the jaws. Fortunately they are rare in children. The

Fig. 37.

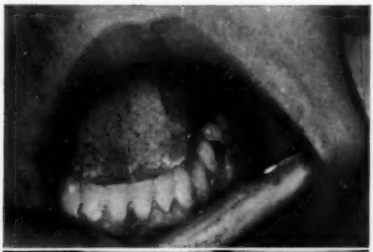

Fig. 38.

Fig. 37. Mandible of a 14 year old boy showing slightly displaced, loose first molar tooth. Biopsy showed metastatic reticulum cell sarcoma not previously diagnosed.
Fig. 38. Radiograph of lower molar region of boy seen in Figure 37. The roots of the second molar are spiked, and the lamina dura is lost.

metastases usually occur late in the course of the disease (Figs. 34, 35, 36), but the primary diagnosis may be made from the jaw lesions (Figs. 37, 38).

SUMMARY

A great variety of benign and malignant tumors may involve the oral regions of young persons. The soft tissues or jaw bones may be the sites of these tumors. Fortunately, metastasis to the jaws is rare.

Because of the relative ease of examination most of these tumors should be discovered early. Thorough oral examination is essential to discover intraoral tumors early. The dentist, through routine dental x-ray examination, may discover insidious intraosseous lesions. Coopera-

tion of the dentist and pediatrician in examination, diagnosis and treatment is one of the best means of assuring reduction in mutilation and death from tumors of the oral regions.

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Ohio State University
Columbus 10, Ohio

TRAUMA IN AND ABOUT THE MOUTH IN CHILDREN

LAWRENCE K. PICKETT, M.D.

DAVID B. STARK, M.D.

Many advances have been made in the treatment of most illnesses and diseases of childhood. The one increasing problem that faces the pediatrician and the surgeon is the treatment of trauma primarily due to accidents. The alarming increase in the accident rate throughout the country and the equally alarming figures in both mortality and morbidity which affect children who have been subjected to trauma point out the need for much more vigorous accident prevention programs throughout the nation. Such figures also point up the need for continuing care and progress in the treatment of trauma. This discussion will be limited to trauma in and about the mouth in infants and children, the special conditions and considerations which arise from such trauma.

SURGICAL AND PROPHYLACTIC CARE

A discussion of accidents in and about the mouth in children will necessarily involve many conditions common to both adults and children. Accepted as standard treatment for both will be good surgical care and prophylaxis against infection. There are, however, some aspects of trauma involving infants and children which require special attention. For many years prime importance has been laid on the immediate treatment of lacerations or other trauma so as to prevent infection. One must not, however, treat the local injury without consideration of the child as a whole, and his general condition.

From the Department of Surgery, State University of New York, Upstate Medical Center at Syracuse.

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In the interests of expediency and good care, often the surgeon involved in repairing trauma would prefer to administer a general anesthetic to a child because of difficult exposure or the expected inability to cooperate. Herein lies an important feature in the treatment of the child. With the onset of trauma and sudden stress the gastrointestinal tract will have a sudden decrease in motility. It is commonly found that food in the stomach at the time of injury may remain there for six to eight hours.

The danger of aspiration of such retained contents certainly must be kept foremost in the mind of the surgeon and the anesthetist anticipating general anesthesia. In the case of elective surgery one is content to allow a period of fasting for six to eight hours, although generally 10 to 12 hours are allowed to elapse. With the stress and urgency of repairing trauma one is inclined to cut this time much shorter, and thus should be all the more alert to the possibility of aspiration of ingested food. To prevent such an aspiration it is urgent that one take a detailed history of the ingestion of food, and, when there is reasonable doubt, resort to gastric lavage for preparation of the stomach if a waiting period is not feasible. If a large meal has been ingested, it is probably wise to wait 10 to 12 hours before attempting a surgical repair if general anesthesia is deemed necessary. During this period of waiting it would undoubtedly be wise to use some form of sedation to allow for normal gastrointestinal motility to accomplish the aim of an empty stomach before anesthesia. Dire sequelae may result from aspiration, and thus too much attention cannot be paid to this aspect.

In addition to the dangers of aspiration, one must also be alert to and aware of the possibilities of a head injury associated with trauma about the face. Again a careful history and observation, as well as a physical examination, will help to indicate the amount of head injury associated with the facial lesion. The extent of a head injury will become evident only after a sufficient period of observation. The obvious signs of increasing drowsiness or coma following a lucid period, and respiratory depression, should turn one's attention to the head injury, leaving the facial injury as a secondary consideration. It has been the general experience of most neurosurgeons that a 24 hour waiting period is adequate for the manifestation of increasing intracranial pressure.

During the period of waiting one can protect the patient by adequate chemotherapeutic coverage. The results from the repair of a laceration some 24 to 36 hours from the time of accident in many cases are not noticeably different from earlier repairs. Certainly the safety of a general anesthesia at this time is far greater than immediately after the injury. What one sacrifices in the nature of possible infection and tissue edema, one certainly gains in the safety of the procedure. An alterna-

tive to general anesthesia, of course, would be local anesthesia for immediate care, with planned secondary plastic surgery if this procedure under local anesthesia is not adequate.

Anesthetic Considerations

Though it is not in the scope of this discussion to go into the details of pediatric anesthesia, several points relative to good pediatric surgical anesthesia should be stressed. Anxiety, fright and pain, which generally accompany an accident, are not conducive to establishing rapport with a young patient. Preparation of the patient for surgery, as in the case of elective surgery, is generally not possible. However, *adequate preanesthetic medication can and should be given* from the standpoint of decreasing secretions to maintain an adequate airway, and also to reduce to a minimum the psychic trauma of induction without a basal anesthetic. A smooth induction and the resultant easier course during anesthesia are a corollary to the protection of the patient against psychic trauma. It has likewise been impressive to the authors that children with a smooth induction, without the struggle too common to induction, have a much more comfortable postanesthetic course with a minimum of nausea and vomiting. Better results of corrective surgery, particularly about the mouth, will be noted if little or no vomiting occurs and oral suction does not have to be resorted to in the recovery room. One must, therefore, use adequate dosages of barbiturates and narcotics in addition to drying agents prior to operative procedures under general anesthetics.

The actual mode of anesthesia is best left to the anesthetist. The adequate use of a basal anesthetic such as Avertin or rectal pentothal will allow a child to be carried in a light plane with the use of ether insufflation by an ether hook when the operation is in or about the mouth. It is frequently necessary to use endotracheal anesthesia in these cases; indeed, in severe cases an elective tracheotomy prior to surgery and for the benefit of the anesthetist should be strongly considered. This will be referred to later in discussions about specific injuries.

Local Anesthesia. Most of the minor trauma seen is amenable to treatment using local anesthesia. The variables in this regard include the age of the child, the position of the trauma and the state of excitement. Here again the judicious use of sedation with a short waiting period will greatly enhance the chances of an optimal result. The quick-acting barbiturates such as Nembutal are easily and quickly assimilated by rectum if the oral route is not possible. A few minutes spent in preparation and sedation often save much time and effort in restraining a child during the procedure.

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Antibiotic Coverage

Since the long-term result in trauma is often dependent on the absence of infection, every effort should be made to prevent such an infection. With the increasing reports of anaphylactoid reactions to penicillin, it behooves one to study each case individually and to apply chemotherapeutic agents judiciously in the adjunctive treatment of the trauma. Careful cleaning and débridement cannot be forgotten as the best possible way to prevent infection. The mouth, face and areas about the mouth are highly resistant to infection. The contamination of trauma is generally of a mixed type and probably better covered by a broad-spectrum type of antibiotic.

One is always faced with the possibility of tetanus infection following trauma. It is, fortunately, an infrequent occurrence that children have not had their preliminary immunizations, but occasionally one is faced with the element of time in older children. The recent report of Stafford et al. shows that a booster injection of tetanus toxoid is effective in evoking a protective response as long as seven years after a previous inoculation. When a serious doubt exists as to previous immunizations or the time element, one must use tetanus antitoxin to protect the child adequately. The usual precautions in skin testing and ophthalmic testing need only be mentioned. Serum sickness and other unpleasant after-effects of horse serum are more readily treated in this day of steroid therapy than in the past. Several cases of tetanus are still seen each year in the larger centers, and thus such protection should not be overlooked.

Wound Treatment

Many of the injuries in and about the mouth will be treated by the family physician or pediatrician. Scarring resulting from a minor injury may be permanent and can be minimized by scrupulous care at the time of the primary repair. It should be remembered that inadequate or ill-advised primary repair of even a small injury may produce a result which is less satisfactory than would have been the case had the injury not been treated. This is particularly true of surface lacerations. The scars which may result from widely placed, coarse sutures tied under tension and left in place too long can be very deforming. Removal of the resultant broad cross-hatched strip of tissue involved may be difficult or impossible. Such a scar is worse than would have occurred if the laceration had been left untreated.

The prime concern in the treatment of facial injuries is the cosmetic result to be obtained. The general principles of treatment of these injuries are similar to the principles of wound treatment elsewhere in

the body. There are, however, some modifications made necessary by the local considerations.

Cleansing of the wound is important. It is essential to clean the abraded or open surface of all dirt or pigmented matter. If this is not done, the pigment may be retained and the area will be tattooed. The pigment particles are transported into the deeper layers of the cutis, and removal may be difficult and will require a secondary operative procedure. Immediate removal of the dirt with soap and water, using a gauze or a stiff brush, may be the best and more simple procedure. Small abrasions are frequently overlooked, and the resultant cosmetic deformity due to the tattooing will be out of all proportion to the apparent severity of the original injury.

After the surface dirt has been removed the deeper parts of the wound are cleansed. Foreign body particles, loose, unattached bone and fragmented teeth should be removed. The use at this time of abundant saline irrigations is helpful. Because of the excellent blood supply to the region, it should not be necessary to remove as much tissue as in débridement elsewhere. Only the obviously dead tissue is to be sacrificed.

The mucous membrane, when lacerated, should be repaired. A small perforation of the mucosa may be ignored, but any laceration extensive enough to raise a significant flap should be closed. When the area is accessible, as in the anterior portion of the oral cavity, a monofilament nylon suture may be used. In less accessible areas or in a patient who is not likely to cooperate at the time of suture removal (most young children) the use of plain or chromic catgut may be preferable. The tissue reaction about the nylon suture is much less marked.

Fine suture material is used to approximate the subcutaneous tissues in such a manner as to allow accurate approximation of the skin edges without tension. The skin edges may be undermined for 1 to 5 cm. if surface loss is great, in order to aid in this approximation. The dermal sutures should be fine; 5-0 or 6-0 monofilament nylon suture has been found useful. The sutures should be interrupted and placed evenly close together and near the skin margin. Use of the dermal sutures placed near the skin edge should not be followed by noticeable scarring from the stitches if the sutures are removed in four days.

In those cases in which approximation of the skin edges is impossible without the exertion of a dangerous amount of tension or without causing the distortion of facial features, one of the minor one-stage plastic procedures may be resorted to. These procedures are based on the principle of borrowing adjacent cutaneous tissue to fill in the traumatic defect. Z-plasties and rotation or sliding flaps may be used.

The possibility of using a split-thickness graft to cover a residual bare area should be mentioned. In many cases, if the graft takes, it

will form an excellent physiologic dressing and will provide a relatively clean field for the performance of plastic procedures at a later date if they should be needed. *No plastic procedure should be performed unless the surgeon is well acquainted with the use of that procedure and unless it is evident that failure of the procedure will not increase the difficulties of subsequent reconstructive surgery.*

When and how to drain a wound of the face has been and continues to be discussed. Many small wounds can be closed safely without drainage. A more extensive wound, particularly if contamination has been significant, should be drained by a small Penrose drain.

Having reviewed the general principles of wound treatment of the face, mention of some of the problems of treatment of wounds involving specific locations will be made.

THE NOSE

In treating the lacerated nose the problems are the associated nasal deformity and the difficulty of replacing tissue lost. The alae must be repaired accurately, because notching and asymmetry here are easily noticed. Frequently the laceration is associated with injury to the bony and cartilagenous framework of the nose. This malposition may sometimes be corrected and the correction maintained by splinting at the time of repair of soft tissue injury. This will depend not only on the extent of the laceration, but also on the degree of injury to the bony framework of the nose and to the septum.

Tissue loss over the nose is serious because of the difficulties of replacing the cutaneous covering by transplanting local tissue. If the loss is extensive, no attempt at reconstruction should be made at the time of primary treatment. The use of split-thickness skin as a primary dressing should be considered. The cosmetic result of a split-thickness skin graft to the nose is not satisfactory because of an inadequacy of texture and the color match with the surrounding skin. Under ideal circumstances, then, a full-thickness skin graft obtained from behind the ear may be tried with the realization that the chances of successful take are probably far from 100 per cent.

THE LIP

The important point of the repair of any laceration of the lip is the accurate approximation of the mucocutaneous line of the two sides to avoid unsightly notching. Care should be taken in placement of deep sutures to approximate the orbicularis oris muscle properly. If there is moderate loss of tissue from an injury to the lower lip, a **V** excision of the defect may be performed, as in the removal of a carcinoma of the

lower lip. In the presence of more extensive tissue loss, closure may be facilitated by use of the advancement procedure commonly used for the repair after removal of a large carcinoma of the lower lip.

THE CHEEK

The problem of main importance in repairing an injured cheek is the prevention of an external salivary fistula if the parotid duct has been injured. A good deal has been written about the end-to-end anastomosis of Stensen's duct in the immediate repair of injuries to the cheek. Results have not been spectacular. The simplest procedure, which apparently gives as good results as any, is the placing of a drain from the region of the severed duct into the oral cavity. A Penrose drain is held in place by several chromic catgut sutures fixed in the depths of the wound in the vicinity of the severed duct. The drain is left in place until it comes out spontaneously, usually after two to four weeks. By this time a satisfactory internal salivary fistula will have formed. This avoids the formation of an external fistula, which is difficult to eradicate.

A deep laceration of the cheek may also injure one or more branches of the facial nerve. If a paralysis of the facial muscles is noted in association with a recent laceration of the cheek, particular care should be taken at the time of closure of the wound. A stimulator should be used to locate the distal nerve branch. The proximal branch may be located by inspection of the opposing wound surface. If question exists about a suspected proximal nerve branch, a small section may be removed for identification by immediate microscopic examination. After identification of the proximal and distal ends accurate approximation should be obtained, using a fine nonabsorbable suture. Two or three sutures of 6-0 ophthalmic silk placed in the perineurium lightly approximating the nerve ends may allow a satisfactory regeneration.

LACERATIONS WHOLLY WITHIN THE MUCOSA OF THE MOUTH

The most common cause of such lacerations is perforation from a foreign body held in the mouth of the child. The candy stick, pencil or almost any toy may be the damaging factor. As mentioned previously, only those lacerations which have raised a significant flap of tissue require surgical closure. The healing of the mucosa and the resistance to infection are great. The general anesthetic required to allow closure of an intraoral laceration is not justified in the usual small laceration.

Most lacerations of the tongue do not require closure. Bleeding, although at first profuse, will usually control itself. Attempt to suture a small laceration of the tongue when bleeding has spontaneously

stopped will inevitably cause a recurrence of this bleeding. Usually the laceration will heal without significant scarring. When the laceration involves the border of the tongue, a split-tongue or "step-off" type of scar may result which would be a constant source of intraoral annoyance. In this instance closure of the laceration may be indicated. If closure is needed to control bleeding, deeply placed vertical mattress sutures may be effective. If simple approximation is required, buried sutures will be more satisfactory. The annoyance of a suture on the surface of the tongue is evident. Generally the child will remove this annoying foreign material before the healing of the wound is complete. Buried sutures properly placed will provide adequate approximation and will not be annoying to the patient.

An extensive laceration of the tongue or of the floor of the mouth may result in alarming edema to the point of actual respiratory obstruction. Urgent measures may be necessary, and on occasion a tracheotomy will be required to secure a safe airway. Although a liquid diet by mouth is usually possible, feeding by a nasal gastric tube for a few days until the intraoral swelling has subsided may be necessary. A gastrostomy for feeding purposes is seldom necessary, since the edema and discomfort are generally of short duration.

A laceration of the floor of the mouth may be complicated by injury of the submaxillary duct. Complication following this wound is not likely unless the laceration involves also the skin in the submental or submaxillary region. In this instance an external salivary fistula may form. Prevention of this is accomplished by suturing a Penrose drain in the region of the lacerated duct and passing it into the mouth in a similar manner to that described for draining the wound of the cheek, to avoid an external salivary fistula from the parotid duct.

Herniation of the sucking or buccal fat pad following a laceration of the buccal mucosa presents an alarming appearance. It is more common in infants and young children. With the edema subsequent to such a herniation the mouth may be filled with the seemingly well encapsulated mass of adipose tissue. In this situation speed in repair, all else being equal, is of great advantage. Even though there is some doubt as to the viability of the herniated fat, as much as is possible should be replaced and the mucosa should be sutured over the replaced fat, even under some tension. Loss of this fat pad may give rise to a hollow appearance of the cheeks in young children and infants when the pad is particularly well developed. Survival of even a part of the fat pad may prevent a disturbing cosmetic result. Since the buccal fat pad often is in close association with the duct of the parotid gland, allowance should be made for proper placement of opening of the duct in the oral cavity at the time of closure of the mucosal defect.

The complication of spreading infection is of importance with lacera-

tions situated in two areas—the floor of the mouth or the posterior pharynx. With lacerations in these areas prompt and adequate administration of antibiotics should be the rule. The complication of cervical edema with respiratory obstruction or spread of an infection to the mediastinum should be considered and careful observation of the patient's clinical course should be made.

An extensive laceration of the soft palate resulting in actual separation of the halves of the palate should be repaired with care to approximate the muscle groups. Avulsion of mucoperiosteum from the hard palate will ordinarily result in no permanent defect. The tendency for prompt secondary healing of such palatal defects is pronounced.

Hemorrhage occurring as the result of an intraoral laceration may be alarming. Immediate attention should be given to the maintenance of an adequate airway. Usually the bleeding will subside spontaneously if the child is adequately reassured and sedated. Control may be aided by application of pressure to the bleeding area. Occasionally bleeding may continue in spite of sedation and local pressure. This continuing hemorrhage may be an indication for surgical intervention.

BURNS OF THE MOUTH AND OROPHARYNX

Thermal Burns

Most thermal burns about the mouth unassociated with other severe body burns are minor. The mucosal tissues of the mouth are peculiarly capable of rapid repair. Local irrigations may be comforting to the child. The edema from a severe burn inside the mouth and nasopharynx may well result in respiratory obstruction. Delay in development of this edema requires diligent observation for 18 to 24 hours following exposure to heat in an amount such that significant injury may result. If respiratory obstruction occurs, a tracheotomy is indicated. If the thermal insult has been hot air or steam, bronchopulmonary damage may be severe and irreversible.

A nasal gastric tube may be used for feeding. If the damage is severe, a gastrostomy may be performed. It is rare for a hot liquid to cause damage in the esophagus. The cooling of liquid in transit generally prevents actual esophageal burns.

Chemical Burns

Chemical burns are of more serious import. Fortunately, chemical burns occur less frequently now than in past years. The immediate emergency treatment is of great importance. Any dilution of the agent will be helpful. If the nature of the agent is known, neutralization as

quickly as possible is the optimum treatment. For an alkali, vinegar diluted 1:1 will act as an efficient neutralizing agent. Milk may be used to counteract the effects of acid. If a significant amount of the material has been ingested, gastric lavage associated with dilution and emesis will be helpful.

The care following the chemical burn is difficult. Edema, pain and scar contracture are the features to be considered. Pain is the easiest of the triad to treat. Edema should be regarded as in other trauma, as a potential enemy of the airway. A tracheotomy may be required. The effects of scar contracture will depend upon the site of injury. Damage to the lips may result in the formation of a microstoma. Correction of this deformity will require a plastic procedure after the scar has matured and softened. Obstruction of the digestive tract seldom occurs proximal to the esophagus.

A detailed consideration of cicatricial esophageal obstruction is not within the scope of this paper, but early treatment should be considered a part of the treatment of the buccal burns. One must be cautious about attempting to pass a gastric tube down a severely injured esophagus. Hemorrhage or perforation of the esophagus may result from leaving such a tube in place. If possible, the patient should be made to swallow a string. This string may be brought out through the nose and taped to the face. There is little associated discomfort or inconvenience. When the acute reaction has subsided, the esophageal stenosis may be treated by antegrade or retrograde dilatation (assuming for the latter that a gastrostomy has been performed). The presence of a string passing from the pharynx through the scarred esophagus into the stomach makes the procedure of dilatation safer and more efficient.

Electrical Burn of the Mouth

The third type of burn is an electrical burn of the mouth. This injury occurs most commonly in the toddler group. When exploring the floors and spaces behind furniture, they encounter electrical extensions. Commonly the child mouths the female component of the extension. Saliva or, in some older style outlets, the tongue makes the contact, and the house current hits the child.

Occasionally the child will be found to have bitten through the insulation, making the contact directly with the teeth. Electrocution has been reported, but generally only local burns result. The severity of this burn varies with the factors, including the character of the contact and the duration of the exposure. In several observed cases the alternating current resulted in tetanic contractions of the muscles of mastication, causing the teeth to grip the fixture and to hold the contact. The immediate obvious charring and burning of the lips,

tongue and gums seldom represents the full extent of damage. A characteristic feature of electrical burns is the tendency of the destruction of tissue beyond the gross limits of the burn, frequently along the course of nervous and vascular tissues. For this reason the eventual loss of tissue is frequently greater than at first estimated.

Hospitalization is generally required for feeding and local care of the wound. The most alarming complication of this injury is the secondary hemorrhage, which may occur at any time following the fifth day until the slough has separated. This may be difficult to control. Because of this definite danger, hospital observation is usually indicated until complete separation of slough.

The resultant scarring will cause a characteristic defect of the mouth consisting in a microstoma or a circular "whistling" defect at the involved angle of the mouth. With extensive injury to the tongue or the alveolar ridge, late speech defects and dental complications can be anticipated.

FRACTURES OF THE JAW

Fractures of the maxilla or mandible are less in children than in adults who have undergone the same degree of trauma. This may be due to the relatively light body weight which gives a momentum to the child thrown from an automobile. There is a correspondingly lesser degree of force thrust on the face when this strikes first. An alternative explanation is the relatively greater resiliency of young bones because of the increased cartilage content.

Fractures of the Mandible

The diagnosis of a fracture of a mandible can usually be made by clinical examination. The findings may be a palpable irregularity in the mandibular arch, a displacement of the lower teeth so that the teeth do not close in proper occlusion, or a visible or palpable tear of the oral mucosa overlying the alveolus. X-ray examination should be made when a fracture is suspected. The x-ray film will demonstrate the exact site of fracture in the presence or absence of comminution. The commonest site of fracture of the mandible in a child is at or in front of the bicuspid area.

Treatment. If there is no significant displacement, no active treatment is indicated. Comfort may be obtained by the use of a Barton dressing placed about the head, but this is difficult to maintain in position on an active child. The child should be kept on a liquid or soft diet for three to four weeks. At this time usually the union will be firm enough to per-

mit starting a fuller diet which will require some chewing. The fracture will usually be firmly healed by five weeks. If there is significant displacement, some form of fixation of the fragments in proper position must be undertaken. In the older child it may be possible to maintain proper position by wiring of an apparatus to the upper and lower dental arches, which permits drawing the displaced fragment into proper position and maintaining it there while the upper and lower jaws are in occlusion. In the younger child, fixation of such an apparatus is not possible.

One satisfactory method of immobilization of the mandibular fracture in the younger child is the approximation of the mandible to the maxilla using a wire passed circumferentially about the midline of the mandible and fixed to the maxilla through a drill hole placed in the anterior nasal spine. Access to this point is obtained through a small incision in the midline of the upper alveolobuccal sulcus. If the fracture is near the midline, this technique will not be satisfactory. For these children a properly formed silver splint is fitted to the reduced lower jaw and held in place by wires placed circumferentially about the mandible and the splint. This permits the patient to open the mouth. A silver splint is made by first obtaining a plaster impression of the lower jaw in its displaced position. The plaster model is then cut at the line of fracture and the displacement of the fragments is corrected. A silver splint is then made up to fit the teeth, which have been aligned in proper position. It is usually left in place for four weeks, after which it is removed and graded activity is permitted. These two techniques avoid the complication of infection and injury to the teeth buds which may result from open reduction and wiring of the fragments.

Subcondylar Fractures. The second commonest site of fracture of the mandible in a child is the subcondylar area. The incidence of this fracture is fortunately rare. The clinical diagnosis of this fracture may be made by the presence of swelling and pain in the region of the temporal mandibular joint or below, together with the displacement of the lower dental arch and trismus or muscle spasm on the side of the injury.

TREATMENT. If the displacement is minimal, no active treatment is indicated. The child should be allowed only liquids or soft foods. After three weeks standard diet may be gradually used. If the displacement is more definite, the dental arches should be brought into proper position and maintained there by the application of the arch bars in the older child or the circumferential wire about the mandible and fixed to the anterior nasal spine in the younger child. Again fixation and immobilization is maintained for three weeks.

The parents should be informed of the possibility of the development of ankylosis. This complication is much more likely to occur when the

fracture is in a child than when the same anatomic fracture occurs in an adult. There is also the possibility that the growth center in the region of the mandibular head will be interfered with, so that there will be a lack of subsequent growth of the mandible. This lack of development will result in an asymmetry of the face. This lack of growth is less likely to occur in the older child. Open reductions and fixation of subcondylar fractures, although advocated by some, do not appear justified or desirable. The incidence of absorption of the head following such manipulation is significant. This absorption will of course be associated with the lack of vertical growth. Similarly, even though absorption does not occur, the manipulation necessary may result in injury to a growth center which otherwise might continue to function satisfactorily.

Fractures of the Maxilla

Extensive fracture of the maxilla in the child is uncommon. The most frequent form of injury involves fracture of the alveolar ridge with displacement of the teeth. The care of these teeth is discussed in another article in this issue (p. 979). The clinical diagnosis of this injury is by inspection, demonstrating displaced teeth, and then by palpation demonstrating the displacement and mobility of the fragments. More extensive maxillary fractures, although uncommon, may be treated as for the adult. The clinical diagnosis is by observation of a contoural deformity of the face or by palpation of a bony asymmetry of the face or mobility of the maxilla. Swelling of the soft tissues with ecchymosis of the eyelids and the conjunctiva is characteristic.

The presence of extensive fractures of the maxilla, particularly in the child, is associated usually with a head injury. Consideration of the head injury takes priority.

Treatment. A depressed malar zygomatic component may be corrected within the first four days by simple elevation. If the fragments are comminuted or if the fracture is of longer duration, elevation will be accomplished by entering the antrum and maintaining reduction by packing. The bony structures and antra are small in children, and so adequate reduction may be more difficult than in adults. If there is a bilateral fracture of the maxilla with mobility of the upper jaw, the treatment may consist in maintenance of proper occlusion in the manner discussed under treatment of mandibular fractures.

The complication of lack of subsequent normal growth of any part which is seriously traumatized must be considered. The parents should be informed of this possibility at the time of treatment. Unless the fracture is severe and the fragments are markedly comminuted so that reduction is not possible, subsequent development is usually adequate.

Associated Complications

Hemorrhage. Fractures of the upper or lower jaws which result in tearing of the mucous membrane will result in bleeding. Ordinarily this bleeding is not of significance; however, it may be profuse. The main danger of this bleeding is the resultant filling of the airway, producing respiratory obstruction.

Respiratory Obstruction. Patients with fractures of the upper and lower jaws may present themselves with subacute or acute respiratory obstruction. This obstruction may be due to bleeding into the oral cavity, to swelling of the soft tissues due to trauma, or to inadequate support of the tongue and soft tissues because of a break of the mandibular arch. Further obstruction may result from a collection of saliva and mucus in the pharynx which cannot be swallowed because of pain, swelling or disruption of the swallowing mechanism. This respiratory obstruction is much more likely to occur if there is an associated head injury which reduces the level of consciousness of the patient.

When respiratory obstruction is evident for one or more of the reasons enumerated above, an early tracheotomy is indicated and may save the life of a patient who would otherwise die of acute asphyxia or of a bronchopneumonia secondary to prolonged borderline respiratory obstruction and aspiration.

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713 East Genesee Street
Syracuse 3, New York

CLINICAL PROBLEMS

RELATED TO THE TONGUE

E. CHERASKIN, M.D., D.M.D.

ROBERT T. BINFORD, JR., D.M.D.

In the physical examination of the head and neck the oral cavity too frequently receives cursory consideration. The purpose of this clinic is to present an outline of the many local and systemic diseases which may affect one region—the *tongue*. The fact that numerous generalized diseases are mirrored in various lingual changes is generally appreciated. It is hoped that the reader may glean from this survey a practical concept of the importance of an examination of this organ. In striving for comprehensiveness it has been necessary to limit the detail devoted to any specific condition. Many excellent articles are available which describe fully the clinical and pathologic features of the diseases considered in these pages.

Few of the lingual signs are pathognomonic. However, in the constellation with other historical and physical findings the tongue changes will often "cinch" the diagnosis.

In actual clinical practice the child does not present himself with a disease. Actually, the examiner is provided with a series of symptoms and signs. The outline in the following pages is designed to aid the clinician in organizing his observations so as to arrive at a logical diagnosis.

ANATOMY AND PHYSIOLOGY OF THE TONGUE

Anatomy

In order to understand lingual pathology it is necessary to consider a few of the clinically important anatomic features.

The tongue is a highly vascular and muscular organ occupying most of the floor of the mouth and forming the anterior wall of the oro-

From the Department of Oral Medicine, University of Alabama School of Dentistry, Birmingham.

pharynx. The dorsum is divided into an anterior two thirds and a posterior third by a V-shaped groove, the *sulcus terminalis*. The dorsum in the relaxed tongue lies just below the occlusal level of the lower teeth. The position of the tongue can be variable and is of importance, as we shall see, in tongue habits. Embryologically, the tongue is formed from the fusion of the first, second and third branchial arches. Persistence of the tuberculum impar, which should normally recede as the two halves of the tongue fuse, results in a congenital lesion called median rhomboid glossitis.

The squamous epithelium covering the dorsum is quite specialized and unlike the surface epithelium of other mucosal tissues. On the anterior two thirds three types of papillae are recognized: (1) *filiform*, (2) *fungiform* and (3) *circumvallate*. The *filiform* papillae contribute to the tongue its velvet-like quality. They are thin, jagged, epithelial projections, between 1 and 3 mm. in length, and are generally arranged in rows. These papillae undergo continual desquamation and regeneration. Their presence or absence contributes greatly to lingual coating and smooth tongue. In children the filiform papillae are not as pronounced as in the adult. The *fungiform* papillae are interspersed among the filiform ones, but are more numerous along the side and tip of the tongue. They are mushroom-shaped, larger, and are readily distinguished by their redness. They are not as sensitively attuned to metabolic changes as the filiform type. The *circumvallate* papillae, numbering eight to ten, are arranged in a V-shaped line just anterior to the sulcus terminalis. They are large and flat and are surrounded by a groove containing serous glands and taste buds. The foramen cecum is located at the apex of the V. This represents the embryologic vestige of the thyroglossal duct—a remnant of the early development of the thyroid gland.

The posterior third of the tongue is not readily inspected in the routine examination, nor is it particularly important in disease. Suffice it to say that its surface contains lymphoid follicles, collectively referred to as the lingual tonsil.

The general sensory innervation to the anterior two thirds of the tongue is mediated by the lingual nerve and the gustatory sense by the chorda tympani branch of the facial nerve. Both general and taste sensation of the posterior third is served principally by the glossopharyngeal nerve and to a minor degree by the vagus. Motor innervation to the entire organ is made possible largely by the hypoglossal nerve.

Physiology

The tongue subserves four functions: (1) *taste*, (2) *speech*, (3) *mastication* and (4) *deglutition*.

Taste. The taste buds are to be found in the circumvallate papillae and to some extent in the fungiform papillae. There are taste receptors, however, in other parts of the oral cavity. Though no anatomic support can be found, it is probable that there are four types of taste receptors for sweet, sour, bitter and salt.

Speech. The phonatory importance of the tongue is readily evident from the observation that the production of many consonants depends upon the proper positioning of the tongue with respect to the teeth and palate.

Mastication. It is clear that mastication is a complex process. In order for food to be divided and macerated, it must be tossed upon the broad grinding surfaces of the posterior teeth. This is made possible by the extraordinary mobility of the tongue.

Deglutition. Deglutition may be divided into three stages. The first, the only voluntary phase, is made possible when the posterior portion of the tongue containing the bolus of food is depressed while the apex is elevated. This act hurls the bolus into the pharynx, and the second stage of deglutition begins.

CLINICAL EXAMINATION OF THE TONGUE

Methods of Investigation

The tongue can be examined by (1) *inspection,* (2) *palpation,* (3) *with the aid of a pharyngeal mirror* and (4) *Wood's light.*

Fortunately, most pathologic signs can either be seen or palpated. The protruded anterior portion of the tongue is easily visualized. The lateral borders are best seen if the tip of the tongue is held with gauze and gently retracted to the right and left. The lateral edges are a frequent site for traumatic lesions, and when posteriorly located they may otherwise easily escape notice. A tongue blade and a pharyngeal mirror are useful for visualizing the posterior aspect. Bimanual palpation is desirable for examining the corpus of the tongue and the sublingual space. This is best accomplished with the tongue in a relaxed position.

Recently there has been a renewed interest in the possible use of Wood's light (filtered ultraviolet) as an intraoral diagnostic aid. When the mouth is flooded with filtered ultraviolet light, excluding the visible rays, the dorsum of the tongue often displays a red-orange fluorescence believed to be due to the presence of porphyrins. In the majority of healthy, well nourished people the fluorescent area extends either over the entire dorsum or at least its anterior half. It has been observed that with papillary atrophy, as is seen in the so-called smooth tongue, there is an absence of this fluorescence. That the various vitamin B fractions seem to influence the restoration of fluorescence is doubtless due to

their action in regenerating the papillae. There is, however, no absolute proof at present that reduced or absent fluorescence is always due to a vitamin deficiency. Fluorescence seems to decrease with age even though there is no obvious disease or malnutrition.

Symptoms

By definition, a symptom is *subjective*, and pain is the most common complaint.

Pain. The most important cause for pain is mechanical trauma to the tongue. This situation develops with accidental biting and under a host of other traumatic conditions. Probably, next in frequency, are systemic and local infective states leading to vesicle and ulcer formation (e.g., herpetic gingivostomatitis). In some cases lingual pain is described as soreness or burning. When this symptom accompanies papillary atrophy, erythema and enlargement, a *deficiency glossitis* should be suspected. Such a clinical picture may result singly or in combination from microcytic hypochromic anemia, macrocytic hyperchromic anemia, and vitamin B deficiency states. These diseases may in turn be related to gastrointestinal disturbances. A protein deficiency, particularly of tryptophane, may induce such a glossitis. Since the atrophic changes are not pathognomonic, blood studies, gastric analysis and an adequate dietary history are necessary to make a diagnosis. In addition, a thorough physical examination should be directed toward finding other confirmatory evidence, such as angular cheilosis, dermatitis and lassitude. It is rare in children to find lingual pain with no organic changes. In contrast, this does occur frequently in climacteric women, in whom various oral complaints are the result of psychogenic causes.

The presence of pain becomes important in differentiating deficiency glossitis from geographic tongue, median rhomboid glossitis, black hairy tongue and a host of other conditions.

Other Symptoms. Important information is sometimes obtained from other presenting complaints. Fusospirochetosis frequently produces a metallic taste. However, true abnormalities in the gustatory sense are exceedingly rare. Difficulty with speech and faulty articulation may be associated with macroglossia, hysteria and various neurologic disorders.

Signs

By definition, a *sign* is an *objective* finding. The examination of the tongue should include an appraisal of the following: (1) *tongue size*, (2) *position* and *mobility*, (3) *color*, (4) *coatings*, (5) *primary* and

(6) *secondary dermatologic lesions,* (7) *papillary status,* (8) *grooves,* (9) *scars,* and (10) *malformations.*

Tongue Size. In the final analysis there are only *three* possibilities: (1) *enlarged tongue,* (2) *normal tongue,* (3) *small* or *absent tongue.* Tongue enlargement is by far the most common pathologic finding.

MACROGLOSSIA. This condition, also known as *enlarged tongue,* may be (1) *generalized* or (2) *localized* in its distribution. Thus the entire tongue may be involved, or only one portion. For example, congenital muscular macroglossia is of the generalized type. In contradistinction, infection, simple mechanical trauma or a neoplasm produces enlargement of only a segment of the tongue. Macroglossia may also be (1) *transient* or (2) *permanent* in its clinical course. Thus infectious glossitis, for example, produces swelling for a limited time. In contrast, developmental and untreated hormonal disturbances yield a more or less permanent macroglossia. Tongue enlargement may be of (1) *congenital* or (2) *acquired* origin. Thus the infant may be born with a large tongue, or this condition may arise postnatally. Hemangiomas and lymphangiomas are relatively common examples of congenital neoplasms which lead to localized enlargement. And, finally, macroglossia may be (1) *absolute* or (2) *relative.* Thus there may be a true enlargement. This can be traced to an increase in cellular (e.g., rhabdomyoma) or extracellular (e.g., edema) factors. On the other hand, the tongue may only appear large because the neighboring structures are small. In other words, the tongue seems large with respect to a small mouth. A relative macroglossia is observed in the young, healthy infant. It is also encountered in mongolism. It should, therefore, be clear that the presence of macroglossia demands an answer as to whether the problem is (1) *generalized,* (2) *localized,* (3) *transient,* (4) *permanent,* (5) *congenital,* (6) *acquired,* (7) *absolute* or (8) *relative.* Obviously, the solution must be gleaned from the history and the rest of the physical examination.

The diseases of pediatric interest which produce macroglossia are classified in Table 1.

MICROGLOSSIA AND AGLOSSIA. A small tongue (*microglossia*) and its total absence (*aglossia*) are indeed rare. Usually, in children, a reduced or absent lingual organ is of congenital origin.

Tongue Position and Mobility. The physiologic position of the tongue has already been considered (p. 920). It should be clear that the tongue can assume only a limited number of abnormal positions: (1) *elevation,* (2) *glossoptosis* or (3) *forward thrusting.* As already pointed out (p. 921), the tongue is capable of a great range of motion. Any consideration of tongue pathology must necessarily include a discussion of (1) *tongue elevation,* (2) *glossoptosis,* (3) *hypermobility* and (4) *hypomobility.*

ELEVATION OF THE TONGUE. This common finding is generally associated with infections and particularly those which develop in the sublingual space. Similarly, any neoplasm or cyst in this region can cause such displacement.

GLOSSOPTOSIS. In this condition the tongue is displaced in a posterior direction possibly to the point of respiratory obstruction. It may be seen in congenital mandibular hypoplasia.

TABLE 1. *Diseases Which Produce Macroglossia*

INFECTIOUS DISEASES	CYSTS
Actinomycosis	Mucocele
Histoplasmosis	Thyroglossal duct cyst
Ludwig's angina	HORMONAL DISTURBANCES
Smallpox	Cretinism
Thrush	Juvenile myxedema
Tuberculosis	Diabetes mellitus
Nonspecific glossitis	Hypophysial gigantism
PHYSICAL DISORDERS	DEVELOPMENTAL DISTURBANCES
Foreign bodies	Congenital muscular macroglossia
Jaw fractures	Mongolism
Mechanical edema	Gargoylism
CHEMICAL DISORDERS	NUTRITIONAL DISORDERS
Plumbism	Ariboflavinosis
Wasp and bee stings	Beriberi
NEOPLASMS	Pellagra
Fibroma	Vitamin C deficiency
Hemangioma	STRESS REACTIONS
Lymphangioma	Angioneurotic edema
Lymphosarcoma	Erythema multiforme
Lipoma	Serum sickness
Neurofibroma	Stomatitis venenata
Multiple myeloma	Urticaria
Papilloma	MISCELLANEOUS DISORDERS
Rhabdomyoma	Cardiac decompensation
	Cardiac glycogen disease
	Progressive muscular dystrophy

FORWARD THRUSTING. This habit often follows efforts at the breaking of a previous thumb-sucking habit particularly when the latter has produced an open bite (space between upper and lower anterior teeth). The patient will thrust the tongue into the open space each time he swallows. This action obviously perpetuates the dental deformity. Psychotherapy may be required to correct this habit.

HYPERMOBILITY. This neuromuscular sign is a fairly common finding and is usually related to anxiety. Marked tremors on protrusion, however, frequently suggest Sydenham's chorea. This sign may also be present in chemical intoxications (e.g., plumbism) and also in hyperthyroidism and rheumatic fever.

HYPOMOBILITY. This is invariably significant since under normal conditions the tongue is extremely mobile. A painful glossitis, neurological

disorders which involve the muscles of the tongue, and ankyloglossia (tonguetie) all restrict the range of tongue action. The incidence of lingual hypomobility in cardiac glycogen disease is great and should be borne in mind. Table 2 presents an outline of conditions producing this sign.

Color Changes in the Tongue. Seldom are color changes the only sign of a disease. Alterations in size, papillary status and subjective complaints usually add to the picture. With the exception of the white coated tongue, the redness of inflammatory erythema is the most fre-

TABLE 2. *Diseases Which Produce Hypomobility of the Tongue*

INFECTIOUS DISEASES	STRESS REACTIONS
Bulbar poliomyelitis	Scleroderma
Infective glossitis	MISCELLANEOUS DISORDERS
Sublingual infections	Myasthenia gravis
PHYSICAL DISORDERS	Amyotrophic lateral sclerosis
Ankyloglossia	Myotonia congenita
Traumatic glossitis	Cerebrovascular accident
NEOPLASTIC DISEASES	Bulbar paralysis
Teratoma	Severed hypoglossal nerve
Sublingual neoplasms	Syringomyelia
CYSTS	Cardiac glycogen disease
Thyroglossal duct cyst	
Sublingual cysts	

quent color alteration. Such erythema is either *localized* or *generalized* and may or may not be painful. Usually, papillary atrophy accompanies it.

Localized erythema is generally the result of physical trauma. A badly broken down tooth, an irritating restoration or an ill-fitting appliance may be at fault. Occasionally this accompanies a nonspecific glossitis or, rarely, a lingual abscess. Median rhomboid glossitis is characterized by a smooth, shiny, red, oval plaque located in the midline just anterior to the circumvallate papillae. It is a congenital, benign, asymptomatic lesion. Lingual hemangiomas are characteristically dark bluish-red and are of particular interest in pediatrics because, since they are congenital, they are usually recognized in infancy.

Generalized erythema of the tongue has long been recognized in the various vitamin B complex deficiency diseases. Pellagra, sprue, ariboflavinosis and juvenile pernicious anemia are accompanied by a red and painful glossitis. The pellagrous glossitis is described as beefy red, whereas the tongue of ariboflavinosis is magenta. The diagnosis of these conditions requires considerable supportive evidence in addition to the tongue findings. The nutritional deficiencies are so frequently multiple that it is seldom that a pure syndrome is recognized.

The pallor of the mucous membranes in anemia, the increased redness in polycythemia and hypertension, and the yellow cast seen in

jaundice are further examples of the potential importance of this clinical sign. Diseases which present color changes in the tongue are outlined in Table 3.

Tongue Coatings. For many years investigators have sought to demonstrate a causative relation between coating and other disease processes. It should be remembered that the tongue is frequently coated under physiologic conditions, particularly upon arising in the morning. Such coating results from the passive accumulation of bacteria, mucin,

TABLE 3. *Diseases Which Produce Color Changes in the Tongue**

INFECTIOUS DISEASES	CHEMICAL DISORDERS
Early scarlet fever (W)	Aplastic anemia (R because of purpura)
Late scarlet fever (R)	Sodium perborate (R)
Thrush (R with W curds)	Candies (variable)
Measles (R)	Black hairy tongue (B, Br, Y, G)
Smallpox (R)	NEOPLASTIC DISEASES
Fusospirochetosis (R)	Fibroma (R)
Gonococcal stomatitis (Y)	Hemangioma (R)
Nonspecific glossitis (R)	Papilloma (W)
Secondary syphilis (W)	Lipoma (Y)
Histoplasmosis (W)	Rhabdomyoma (P)
PHYSICAL DISORDERS	Myoblastoma (P)
Galvanism (R)	CYSTS
Traumatic glossitis (R)	Mucocele (Bl)
Traumatic hyperkeratosis (W)	HORMONAL DISTURBANCES
	Diabetes mellitus (W)

* The characteristic color change is indicated in code: *R*, red; *W*, white; *Y*, yellow; *B*, black; *G*, green; *Bl*, blue; *Br*, brown; and *P*, pale.

food debris and desquamated epithelium from the filiform papillae. However, an actual increase in thickness of the papillary epithelium plays a more important role in coat formation than was frequently thought. In support of this is the fact that various dyes painted on the dorsum may require several days before they are removed by the process of desquamation. In healthy persons, chewing, talking and salivation mechanically cleanse the tongue. Thus one finds that the early morning coat disappears after breakfast.

Heavy lingual coatings are observed in children who are habitual mouth breathers or suffer with febrile and other dehydration states. The cause of such lingual coatings is not entirely clear. However, drying of the oral mucosa, lack of mechanical cleansing of the tissues due to nondetergent diets, local hypoxia and regional vasomotor changes all probably serve etiologic roles. A theory of retrograde intestinal flow has been advanced to explain the frequency with which tongue coating and alimentary problems are associated. Attempts to prove this theory have not been successful. Thus materials (lithium carmine, lycopodium) introduced rectally have been recovered from the stomach, but never

from the mouth. Contrary to popular belief, constipation is not associated with tongue coating, although the vomiting of intestinal and pyloric obstructions invariably produces a thick lingual coat. This is found also in severe cardiospasm with esophageal retention. The underlying mechanism is obscure.

Various neurological disorders affecting the hypoglossal nerve may produce coating on the paralyzed side. Kidney disease does not generally affect the tongue with the exception of renal failure. In this condition the coating is brown and is believed to be due to the excretion of nitrogenous products in the saliva. Most generally, the color of the coated tongue is white, but may be changed by foods, tobacco and medicaments.

A summary of the diseases associated with lingual coating is presented in Table 4.

TABLE 4. *Diseases Associated with Lingual Coating*

INFECTIOUS DISEASES	CHEMICAL DISORDERS
Scarlet fever	Agranulocytosis
Diphtheria	Black hairy tongue
Fusospirochetosis	NEOPLASTIC DISEASES
Measles	Leukemia
Typhoid fever	HORMONAL DISTURBANCES
Typhus fever	Diabetes mellitus
Yellow fever	REACTION TO STRESS
Pneumonia	Rheumatic fever
Tonsillitis	Anxiety
Pharyngitis	MISCELLANEOUS DISORDERS
Laryngitis	Gastritis
Acute febrile diseases	Cardiospasm
Psittacosis	Intestinal and pyloric obstruction
PHYSICAL DISORDERS	Renal failure
Traumatic glossitis	
Mouth breathing	
Insufficient mastication of food	
Decreased tongue mobility	
High intake of soft and liquid foods	

Vesicles. There are four primary epithelial lesions: (1) *macules,* (2) *papules,* (3) *vesicles,* and (4) *pustules.* Of this group, the vesicle is the most common lesion observed on the tongue. Broadly speaking, vesicles may occur in connection with viral diseases (e.g., herpes simplex and chickenpox) and thermal trauma, and as a reaction to stress and to antigenic substances (e.g., erythema multiforme). It should be recalled that a vesicle or bulla is thin-walled. Thus, in the presence of the usual intraoral trauma, the vesicle is short-lived. The end result is an ulcer. Thus it is indeed rare to observe vesicle formation anywhere in the oral cavity and especially in a hypermobile and constantly traumatized organ like the tongue.

Ulcerations. There are more than a score of secondary dermatologic lesions. Included in this group are (1) *nodules,* (2) *crusts,* (3) *scabs,* (4) *excoriations,* (5) *wheals,* (6) *fissures* and, most important to this discussion, (7) *ulcers.*

We have already learned that intraoral vesicular lesions rupture promptly and that the end result is an ulcer. By far, however, the most frequent cause of lingual ulcerations in children is mechanical trauma. This may occur from tongue biting or irritation from a sharp edge on a tooth. Ulcerations often follow the ingestion of a hot liquid. It can be observed from Table 5 that there are many disorders accompanied by ulcers on the tongue.

TABLE 5. *Diseases Producing Lingual Ulcerations*

INFECTIOUS DISEASES	PHYSICAL DISORDERS
Fusospirochetosis	Galvanism
Diphtheria	Mechanical and thermal trauma
Syphilis	CHEMICAL DISORDERS
Tularemia	Aplastic anemia
Actinomycosis	Agranulocytosis
Blastomycosis	Simple chemical burn (aspirin)
Histoplasmosis	REACTIONS TO STRESS
Thrush	Urticaria
Herpes simplex	Erythema multiforme
Herpes zoster	Lupus erythematosus
Pertussis	Scleroderma

Papillary Status. The lingual papillae are highly sensitive structures which change in size under a variety of conditions. As a matter of fact, the lingual papillae can actually grow in a matter of days. Basically, there are two pathologic possibilities: (1) *atrophy* of the papillae and (2) papillary *hypertrophy.*

ATROPHY. The smooth or atrophic tongue is always the result of the combined effect of normal or accelerated degeneration and deficient regeneration of the filiform papillae. Although the exact mechanism is not known, tissue respiration appears to be a cardinal factor. Thus a disturbance in the intracellular respiratory enzyme systems is very likely at fault in the riboflavin and niacin deficiency states. The pellagrous tongue is fiery red, edematous and tender. As the condition advances, the filiform papillae atrophy. Tissue hypoxia may also explain the atrophy which occurs in severe cardiac decompensation, and perhaps iron deficiency anemias. It is interesting that both hyperthyroidism and hypothyroidism have been associated with smooth tongues.

Geographic tongue (erythema migrans) occurs not infrequently in children—notably in those suffering with mild febrile diseases. It is a benign, usually asymptomatic lesion characterized by irregular patches of filiform papillary atrophy surrounded by a gray-yellow border. The

atrophic areas migrate continually and therefore produce a "geographic" appearance. The clinical course is indefinite, and the lesions may come and go for varying periods of time from weeks to many years. The fungiform papillae appear prominent because of the contrasting atrophy of the filiform papillae. It is not related to any deficiency diseases, and no therapy is required.

Table 6 schematizes these conditions.

TABLE 6. *Diseases Producing Papillary Atrophy*

INFECTIOUS DISEASES	NUTRITIONAL DISTURBANCES
Syphilis	Ariboflavinosis
Thrush	Pellagra
PHYSICAL DISORDERS	Celiac disease
Traumatic glossitis	DEVELOPMENTAL DISTURBANCES
HORMONAL DISTURBANCES	Geographic tongue
Hyperthyroidism	MISCELLANEOUS DISORDERS
Cretinism	Hypochromic anemia
Juvenile myxedema	Congestive heart failure

HYPERTROPHY. *Black hairy tongue* (lingua nigra) is the only condition which produces enlargement of the *filiform* papillae. It is a rare asymptomatic disorder affecting most frequently young adult males. The papillae may overgrow to the extent that they actually tickle the palate. The pigment depends upon the types of chromogenic organisms trapped between the papillae and is usually black, brown, yellow or green. The incidence of this condition has greatly increased since the advent of antibiotic lozenges. This would suggest an alteration in the balance of normal flora permitting various fungi to flourish. Treatment requires establishment of good oral hygiene and the discontinuance of any topical antibiotic agent. In persistent cases of papillary hypertrophy the topical application of 10 to 15 per cent salicylic acid may be helpful. A 3 per cent hydrogen peroxide mouthwash will remove discoloration, and the application of a 20 per cent aqueous solution of sodium caprylate may be used as a fungicide.

The *fungiform* papillae are sometimes truly enlarged in ariboflavinosis. They give to the tongue a granular appearance. The tongue may be slightly edematous and also show prominent fungiform papillae in thiamine deficiency, although this is not a constant or significant finding. Despite frequent references to the so-called strawberry tongue of scarlet fever, it is seen in only slightly more than half the cases. Such patients show an early white tongue coat with prominent fungiform papillae (white strawberry tongue). Three or four days later the coating disappears and a red beefy swollen tongue results (red strawberry tongue).

Table 7 summarizes these papillary changes.

TABLE 7. *Diseases Producing Fungiform Hypertrophy*

INFECTIOUS DISEASES	HORMONAL DISTURBANCES
Scarlet fever	Diabetes mellitus
Measles	NUTRITIONAL DISEASES
CHEMICAL DISORDERS	Ariboflavinosis
Black hairy tongue (filiform hypertrophy)	Pellagra
	DEVELOPMENTAL DISTURBANCES
	Fissured tongue

Grooves. The median raphe is normally prominent. Usually there are also parallel furrows on both sides of the raphe. When the fissures are irregularly arranged, the term *scrotal tongue* is applied because of the close resemblance to the convoluted pattern of the scrotal skin. Fissuring is generally held to be congenital and may be inherited as an irregular dominant characteristic. The incidence has been judged to be between 0.5 and 5 per cent of the general population. Sometimes congenital fissuring is associated with slight macroglossia, prominent fungiform papillae and geographic tongue. It is of interest that there is a characteristic fissuring of the tongue in mongolism. No treatment is required unless there is irritation present, in which case topical hydrogen peroxide and mild alkaline mouthwashes may be helpful.

Scars. Tongue biting in grand mal epileptic seizures and in nonepileptic infantile convulsions is the most frequent cause of scarring, and preventive measures to guard against this accident should always be taken.

Malformations. Ankyloglossia, fissuring and median rhomboid glossitis constitute the most common malformations. Ankyloglossia (tongue-tie) and fissured tongue have already been mentioned. Median rhomboid glossitis is relatively rare, occurring in approximately 0.25 per cent of the population. The characteristics are specific, and its recognition is simple. Nevertheless, many of these cases have been erroneously diagnosed as a lingual neoplasm. It occurs as a slightly elevated red oval patch in the midline of the dorsum of the tongue and just anterior to the circumvallate papillae. It is devoid of filiform papillae, so that its surface is usually smooth and glistening, although it may present a nodular appearance. This condition is benign, congenital and asymptomatic. Most patients are unaware of its presence, and no treatment is required.

Other malformations include aglossia and bifid or cleft tongue. Their occurrence is exceedingly rare and does not merit discussion. When they do appear, there is usually other evidence of a developmental disturbance in the mandible.

SUMMARY

Pathology of the tongue becomes clinically apparent by way of *symptoms* and *signs*. The most important symptom is *pain*, though there may also be alterations in taste, difficulty in speech or swallowing. Signs of lingual pathology are apparent by changes in *tongue size, position, mobility, color, coatings, dermatologic lesions, papillary status, grooves, scars* and *malformations*. Since no one symptom or sign is pathognomonic, it behooves the examiner to fit together the positive subjective and objective findings. These, then, in combination with an adequate history and physical examination, provide the only means of arriving at an etiologic diagnosis of pathology in the tongue.

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University of Alabama Medical Center
Birmingham 3, Alabama

SALIVARY GLAND INFECTION

JOHN A. BIGLER, M.D.

The salivary gland system is made up of three pairs of glands. The two parotids open into the mouth through Stensen's ducts opposite the upper second molars. The two submaxillary glands have ductal openings beneath the tongue near the frenulum. The two submental glands also have ductal openings beneath the tongue.

The parotid glands secrete a clear serous fluid that does not contain mucin. Frequent infection by way of the duct is undoubtedly safeguarded by the steady flow of saliva. The absence of mucin which is somewhat antibacterial does seem, though, to make the parotids more subject to infection than the submaxillary and sublingual glands, both of which secrete both mucin and serous fluid.

Enlargement of the salivary glands is more commonly due to infection than to any other cause. Any combination from a single gland to all six glands may be involved.

Swelling of the salivary glands may be encountered in the following conditions: (1) parotitis with or without submaxillary and submental gland involvement due to the specific viral infection of mumps; (2) parotitis due to bacterial infection: (*a*) recurrent parotitis, (*b*) suppurative parotitis; (3) infiltration of the glands by tumors such as lymphosarcoma and by leukemic cells; (4) miscellaneous conditions such as enlargement of lymphatics in or adjacent to the glands.

MUMPS

Epidemic parotitis, more commonly called mumps, is a communicable infectious disease. It is a systemic infection with a predilection

From the Children's Memorial Hospital and Northwestern University Medical School, Chicago, Illinois.

for the salivary glands, the pancreas, testicles, ovaries and the central nervous system.

Etiology

Mumps is not as contagious as most of the other communicable diseases. The spread is undoubtedly by contact and droplet infection. Apparently, infected persons may be the source of the infection before there are any signs of the disease. Contacts may develop the disease during the whole period of gland swelling, which may last as long as 14 days. It seems, also, that there may be a subclinical mumps as a source of contagion.

The incubation period is usually between 14 and 21 days.

There is no sex predominance. Mumps is rarely seen under one year of age. It is most common in the four to 15 year age group. Young adults also have the disease, but rarely older people.

Pathology

The parotid glands are most frequently involved. There may or may not be involvement of the other salivary glands. Because death rarely occurs from the disease, pathology reports are uncommon. Weller and Craig[22] did an autopsy on a nine year old child dying of another cause during the time she had mumps. The mumps virus was isolated. There was swelling of the whole parotid with cellular infiltration, mostly lymphocytes, involving the ducts, periductal connective tissue and acini. The changes in the ductal lining varied from edema to complete desquamation of the epithelial cells. Many of the ducts were dilated and choked with cellular debris.

Symptoms and Signs of Parotid Infection

The onset is acute, usually with a feeling of tenseness and then tenderness of the affected gland. The first symptom in young children may be enlargement of the affected gland. Fever may or may not be present. When present, it ranges from 101 to 104° F. The swelling is at first soft, but there is progressive enlargement and firmness. Redness of the skin may occur. There may be a surrounding soft tissue swelling. Fluctuation is never present. Tenderness is always present.

Usually the swelling progresses for two to three days, then remains stationary for two to three days, and then regresses over the next three to four days. It is not unusual to have one parotid affected first, to be followed three to four days later by the other parotid and even the submaxillary and submental glands. At times only the submaxillary

and submental salivary glands are involved. When several glands are involved, there may be swelling and edema over the clavicle and upper sternum.

When parotid gland swelling is present, the ramus of the jaw cannot be normally felt. When the submaxillary glands are involved, they can be palpated as walnut-sized, firm, tender, movable individual glands. The submental glands are difficult to palpate because of the extra glandular swelling.

The absence of swelling and redness of the salivary gland duct openings is of no diagnostic significance. In all cases of parotid gland swelling the duct should be milked. In mumps there is no secretion or a clear secretion, but never a cloudy or purulent one.

The white blood cell count is slightly depressed, normal or slightly increased. It must be remembered that mumps is a systemic infection and that the first symptoms may not be those of salivary gland involvement.

Symptoms and Signs of Systemic Infection

These are less commonly found in the infant and young child in contrast to the older child and young adult.

Central Nervous System. This usually takes the form of a meningoencephalitis. It may precede the salivary gland involvement by one to two days, but often follows it by five to 10 days. There is a rise in temperature, headache, often vomiting, possibly a convulsion and a stiff neck and spine. The cerebrospinal fluid is under increased pressure, the protein is increased, the sugar may be, and the fluid is clear to slightly cloudy, depending upon a cell count of under 25 to 1000, mostly lymphocytes.

Pancreas. There is a rise in temperature, vomiting, pain in the upper abdomen and varying degrees of rigidity. This rarely precedes, but may follow, the salivary involvement by five to 14 days.

Testes and Ovaries. Orchitis and oophoritis are rarely if ever seen before puberty. Such involvement may occur before the salivary gland swelling, but usually follows by five to eight days the parotid swelling. It may be a cause of sterility.

Other rare complications reported have been mastitis, myocarditis, myelitis and deafness.

Differential Diagnosis

Recurrent parotitis is usually easily differentiated from mumps if one massages Stensen's duct. If a milky fluid is obtained, then mumps is not present.

Suppurative parotitis is most commonly encountered in the newborn

and in the aged, but may occur in children. The fluctuation of abscess formation usually is present.

Zygomatic mastoiditis may be differentiated, but in such cases the swelling does not have the parotid distribution.

Adenitis and *cellulitis* may be confusing, but usually in such conditions the ramus of the jaw is normally palpable.

Immunity

One attack usually confers immunity for life even though only one parotid has been involved. Recurrent infections of the parotid glands are rarely due to the mumps virus.

The immune reaction of mumps has been reported by Enders et al.[6, 7, 8]

The "S" or soluble antigen complement fixation titer rises rapidly during the course of the disease and falls off sharply after the first week. The "V" or virus antigen appears after the first week and reaches its peak in about three weeks and then decreases, but a detectable titer always remains.

A skin test has also been developed, but is of value only in retrospect. It is a delayed tuberculin-like reaction obtained with mumps virus antigen. A positive reaction is believed to indicate previous mumps infection.[8, 10]

Prognosis

The prognosis is invariably good.

Prevention

It is only rarely that prevention of infection should be attempted in children before puberty. Thirty to 50 cc. of convalescent serum or 5 cc. of fractionated gamma globulin from the same source may prevent or modify the infection if given during the first week of exposure.[6] Ordinary gamma globulin (that used for measles prophylaxis) is of no value.

Active protection[4, 12, 13] now seems feasible, but is not advocated for children. One to 2 cc. of a suspension of killed mumps virus is used. The number of injections has not been determined. As the virus is grown in egg embryos, its use in egg-sensitized patients may be hazardous.

Treatment

Treatment is purely symptomatic. The sulfonamides and antibiotics are of no value.

Complete bed rest is advisable during the period of gland swelling. Adequate fluids should be given and what foods the patient can take. Hot or cold applications as the patient prefers may give some comfort. Sedation is rarely necessary in the child.

There is still divergence of opinion as to the use of stilbesterol as a prophylactic and therapeutic aid in orchitis. It can be tried in doses of 3 to 5 mg. once or twice daily.[14] In adult males some benefit may be expected from large doses of up to 200 cc. of mumps convalescent serum or 20 cc. of gamma globulin fractions from such serum.[11, 19]

PAROTITIS DUE TO BACTERIAL INFECTION

RECURRENT PAROTITIS

Recurrent parotitis occurs as a swelling of one or both parotid glands, indistinguishable from mumps except that it recurs and that a flocculent material containing pus cells and bacteria can usually be expressed from Stensen's duct. Many such cases have been reported, and it can now be established that it is not recurrent mumps.[2, 9, 15, 17, 18, 20] The age spread is from infants under one year of age into young adult life. As the infection is rather benign and from outward appearances is indistinguishable from mumps, it is not uncommon to have a child experience one or more attacks before the family seeks medical aid for an explanation.

Etiology

The etiology of recurrent parotitis is still obscure. It does not seem to be caused by the mumps virus. Florman[9] has expressed the opinion that it follows a virus mumps infection. To support this theory all cases should have a positive complement fixation and skin tests as well as involvement of salivary glands other than the parotids. Such has not been the case.

In our experience recurrent parotitis has been primary in otherwise healthy infants and children. There seems to be no relation to throat infection, the presence or absence of the tonsils or the condition of the gums or teeth. It occurs in both the healthy and the malnourished infant. In our cases there was usually no history of mumps prior to the onset. Some cases were observed at the time of the first swelling, and epidemic mumps could be safely excluded. In other cases it seemed from the history that the first attack of parotitis was due to a specific viral infection because other members of the family had parotid swellings.

Pearson[18] reported 17 children with recurrent parotitis, bacterial

infection being present in six and no infection in 11. He also found a history of allergy in 11 of the 17 cases. Such a history and findings of allergy have not been substantiated by others.

In all our cases *Streptococcus viridans* with or without other organisms was cultured from the secretion from Stensen's duct. Other organisms have been staphylococcus, pneumococcus, streptococcus and *E. coli.*

It would seem that the infection is an ascending one from the mouth through Stensen's duct. This is borne out by the work of Berndt, Buck and von Buxton.[1] Using a hemolytic *Staphylococcus aureus,* they infected Stensen's duct in 17 dogs and produced parotitis in seven, while parotitis developed in only three of 15 dogs when an artery to the parotid was injected.

Whether infection is the primary cause of the parotitis or whether it is secondary to salivary stasis is not known. Dilatation of the ductal system has been found by sialograms in some cases without evidence of obstruction. Undoubtedly some degree of dilatation must be present to account for the increased amount of secretion that can be milked from the duct.

Pathology

Florman[9] has reported on a biopsy of the parotid in a case of recurrent parotitis. No mumps virus was recovered by inoculation of chick embryos. There was evidence of acute and chronic nonspecific inflammation with slight dilatation of the ducts. There were changes in the ductal epithelium as well as periductal inflammatory reaction. He reports that these are not unlike the findings in mumps.

Symptoms and Signs

The onset is usually acute. There is no sex difference, and the age at the first attack varies from six to eight months up into young adult life. The onset is most common between the ages of four and five.

Swelling of the parotids is sudden in onset as it is in mumps, and it is usually the first noticeable symptom. The whole gland is involved in nearly all cases. The swelling is rarely as large as it often becomes in mumps, and it has a nodular firm feel with little if any edema. Redness of the skin may occur. The swelling may be bilateral or unilateral, and recurrences may involve one side and later the other side. Usually recurrences involve the same gland each time. Fluctuation is rare. The maximum lymph nodes are not enlarged. Though the submaxillary and sublingual salivary glands are frequently involved in mumps, they are never involved in recurrent parotitis.

Stensen's duct opening is usually red and edematous in contrast to

our usual finding in mumps. The expression of pus from the duct is diagnostic. The pus has a characteristic appearance. It is grayish or whitish and is flocculent with flecks and clumps of pus cells mixed in the saliva. We have never observed a bloody discharge. The purulent material is expressed easily from the duct opening by pressure over the affected parotid and then milking the duct along the cheek. Ordinarily one does not see pus coming from the duct without making this pressure. Often it will spurt from the duct opening. Usually only a small amount can be expressed at any one time, and further milking will not produce more for a few hours. This may mean that the duct dilatation cannot be great. We have never been able to express any plugs which might have blocked the duct. In some cases purulent material does not appear until more than 24 hours after onset. We have never found eosinophils in the secretion. It is only rarely that purulent secretion cannot be obtained.

In mild cases the duration of the swelling may be only for two to three days. In the average case it will last for 10 to 21 days, but it is not unusual to have some swelling for several weeks. In the latter cases there will be fluctuations in the size of the gland during that period. When the swelling has been present for more than two weeks, the glands become more nodular and firm and resolution is slow.

The number of *recurrences* varies widely in the individual case. In some there is only one episode of swelling, while in others there may be 20 or more recurrences. These may occur after only a few days, a week or a month, sometimes two to three times a year, and at times after several years. The longer the interval between attacks, the less likelihood there is of a recurrence. The severity of the swelling and the length of time it is present have no bearing upon the number of attacks or the interval between them. It has seemed to us, after observing well over 100 patients, that the younger the age at onset, the more frequent the attacks.

Fever may or may not be present. When present, the temperature varies between 100 and 104° F. rectally. Fever is usually present at the onset, at which time it is highest and persists for only a few days. After that there may be no fever or only a low grade one.

Pain in varying degree is nearly always present. At times it occurs before the swelling. It is rarely severe, and most children pay no attention to it after the first few days. The amount of tenderness varies a good deal, but is rarely severe.

Toxicity is rarely present even when the fever is high and there is bilateral swelling. The children do not appear ill, and it is difficult to keep them in bed. There is little or no difficulty in eating.

In respect to the *blood cell count*, there is usually some leukocytosis of between 8000 and 18,000 with an increase in the polymorphonuclear

cells. As is usual of infections in infancy, the lymphocyte ratio may be high in contrast to that normally seen after the infancy period.

X-ray examinations of the parotids have always been reported as normal. Sialograms, made by injecting contrast material into Stensen's duct, are reported often to show some slight ductal dilatation.

The infection does not seem to be a general one, but is localized to the parotids. We have never seen involvement of the other salivary glands, the testicles, ovaries, central nervous system or other organs. No instance of contagion from recurrent parotitis has been observed.

Treatment

Treatment so far has not been too satisfactory in shortening the course of individual attacks or in preventing recurrences. The sulfonamides are of no apparent benefit. Penicillin and the other antibiotics may shorten the attacks somewhat.

Aspirin is given for fever and comfort. Sedatives such as phenobarbital may be used, but are rarely indicated. Heat or cold applied to the swollen gland may give some comfort, but does not influence the course of the disease. Bed rest should be insisted upon, at least during the febrile period. Any food that is tolerated should be given.

We have used x-ray therapy in a number of cases, and it has seemed to shorten the period of swelling. Frequently the recurrences are decreased in number as the period between recurrences is lengthened. In one case we observed with several attacks of bilateral swelling, x-ray treatment was given to one side. The next two recurrences developed on the untreated side. Treatment was then advised on the other side, and no further attacks occurred during the next year of observation.

It has never been necessary in our experience to resort to surgical intervention by either slitting the duct opening or incising the gland. In fact, such a procedure should be frowned upon until there is sufficient evidence that frank suppuration has occurred and other methods of therapy have failed.

Recurrent parotitis is usually a benign condition that occurs in otherwise healthy infants and children. This is in contrast to suppurative parotitis with abscess formation, whether it occurs in the newborn or is secondary to some other infections or as a complication of surgery, in which case surgical incision may become necessary.

SUPPURATIVE PAROTITIS

Suppurative parotitis as described here includes only those cases in which there is frank or impending abscess formation of the gland. This is in contrast to recurrent parotitis in which purulent material may be milked from the duct, but there is no apparent abscess. We have

observed four children with abscess formation, but in none of them was there a history of previous recurrent parotitis, and in none of them did recurrent swelling of the gland occur after the attack with suppuration.

Florman[9] reports the case of a five and a half year old boy who, it was stated, had mumps at the age of two years. He recovered, but had some residual painless swelling in the right parotid area which enlarged and regressed from time to time. Two weeks prior to admission he was hit by a fist in this area, and shortly afterwards there was marked pain and swelling. Fluctuation occurred, and incision and drainage were performed. *Neisseria catarrhalis* was cultured. Trauma may have been the cause of the suppuration in this case.

Suppurative parotitis is fairly rare. It occurs in the newborn, during childhood and in the aged. Although the mortality used to be reported as from 30 to 45 per cent,[3] it is now much lower since the advent of the sulfonamides and antibiotics. Sanford[21] reported five cases in newborns treated with sulfonamides and incision for drainage in four cases and with recovery in all five.

The onset is acute with fever and swelling of the affected gland. The involvement is usually unilateral, but it may be bilateral. Trauma to the parotid may be a factor even in the newborn in the process of delivery. The skin over the gland becomes red and hot, and soon fluctuation occurs. In the cases we have seen pus could be milked from Stensen's duct. In older children we have seen it occur with an otitis media. The infectious organism is usually *Staphylococcus aureus* and *Streptococcus viridans*, but any organism may be the infectious agent.

Treatment consists in hot applications and the use of penicillin and the broad-spectrum antibiotics. Incision and drainage is usually necessary, but is best delayed until there is a good area of fluctuation.

MISCELLANEOUS SWELLINGS OF THE SALIVARY GLANDS

Calculi in the salivary glands or ducts are extremely rare in children, but do occur in adults.

In *Mikulicz's syndrome* there is swelling of the parotids usually due to leukemia, lymphosarcoma, tuberculosis or syphilis. Blood and bone marrow studies as well as lymph gland biopsy will establish the diagnosis.

In *cat scratch fever* with involvement of the conjunctiva or a face lesion there may be nodular swellings in the parotid area due to lymphadenitis. *Tuberculosis* may also involve these glands. With such swelling there is not a typical parotid distribution. *Zygomatic mastoiditis* may cause preauricular swelling, but it does not go down over the ramus of the jaw.

Lymphangioma or *hygroma* may occur in the salivary gland areas, but because of the type of swelling, the chronicity and the fact that they are often congenital, a proper diagnosis should not be difficult.

Cervical adenitis and the swelling of an *apical abscess* in the submaxillary region should not be confused with salivary gland infection. Palpation of the ramus of the jaw will usually differentiate an adenitis because it cannot be palpated in parotitis. The swelling of an apical abscess is usually firm and indurated and fairly well fixed to the mandible in contrast to the softer and more movable swelling of the submaxillary salivary glands.

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707 Fullerton Avenue
Chicago 14, Illinois

THE PEDIATRICIAN'S RESPONSIBILITY IN DENTAL EXAMINATION

PAUL K. LOSCH, D.D.S.

WARREN R. SISSON, M.D.

It is generally recognized that physicians caring for the health of children neglect dental diagnoses and manifest little interest in dental treatment because of inadequate instruction in this field. The purpose of this discussion is to offer a procedure for dental examination and to make certain comments concerning selected dental conditions with the hope that further interest and study in this important area of child health will be promoted.

THE DENTAL EXAMINATION

After a long experience of clinical cooperation with pediatricians and dentists it is felt that those interested in the care of children should be equipped and able to make an adequate dental examination. The casual oral examination with tongue blade and flashlight is not satisfactory. Often in transcripts of general physical examination we see, under the heading "Mouth," notes of extensive "pyorrhea" where only a mild inflammation of the gum exists because of the presence of food impactions around the necks of the teeth due to poor hygiene. Or again, we see noted "Much dental caries" when all that exists are extensive stains in caries-free dentitions. Little more than a dental mirror and explorer with an adequate source of light such as that provided by a head mirror need be added to the armamentarium. With very little practice these instruments can aid the examiner in making a detailed examination of the teeth. With even a limited knowledge of what to look for the pediatrician can offer timely information and advice to parents as well as prescribe needed therapy.

From the neonatal period through all the periods of development for which the pediatrician holds himself responsible these examinations should be routine.

Examination of the Newborn*

The newborn, of course, offer little for dental observation with the rare exception of *precocious eruption of teeth*—particularly the inferior primary central incisors. These precociously erupted teeth are usually amorphic and of poor quality and often so tenuous in their attachment that, for fear of unobserved abortion and aspiration, they should be removed. If, however, they seem reasonably firm with a reliable fibrous-like attachment, they should be allowed to remain. They rarely interfere with feeding, but sometimes do irritate the tongue to the point that removal is advisable. This situation, whether allowed to remain or not, creates a problem in development which should come to the early attention of a dentist.

On rare occasions *osteomyelitis of the maxilla* at the premaxillary suture may be observed in the newborn. This condition is characterized by marked redness and swelling of the gingival mucosa in this area and, if not treated promptly, may involve the whole facial hemisphere, including the orbit. The usual treatment for osteomyelitis is, of course, promptly instituted.

In some localities there has been a transient vogue of surgical intervention with the *superior frenum labiorum* when it seemed large and seemed to interfere with mobility of the lip. Part of the premise for surgery is that the frenum muscle will extend its attachment into the palatal area and, thus, during subsequent dental development create a wide diastema between the central incisors. The writers frown upon both therapy and concept, since there is no good evidence to indicate this procedure. A simple inquiry into familial tendency toward central diastemata is usually sufficient to confirm the differential diagnosis and forbid surgery.

Examination of Infants

Within normal limits from the fourth to twelfth postnatal month, a more or less orderly eruption of primary teeth begins which is usually completed by the twenty-fourth to fortieth months. These wide differences in chronological sequence can be regarded as within normal limits, and on rare occasions an even wider divergence may be observed, the host ultimately achieving a normally functioning dentition.

* See also page 847.

Tooth Eruption

The complications reflected in the dental apparatus by general growth interference such as may be observed in endocrinopathies will not be discussed here. There are two observations on this point we wish to emphasize. First, under otherwise normal conditions the chronological sequence of dental development may be expected to be patent. In other words, the early or late appearance of the primary teeth predicts the same behavior during the entire dental development. Second, in no instance should generalization be made concerning the total development of the child from observations on his dental development alone. For example, a child sustaining a tumor of the adrenal cortex may have an accelerated linear and sexual development without change in his dental development. On the other hand, a true cretin will demonstrate retardation in all areas of developmental assay. At the same time, from all known means of assay an otherwise perfectly normal child may exhibit precocious maturation in all areas, including dental development.

There are innumerable charts and tables of so-called normal dental development which the practitioner should consult, but always with the understanding that such tables are the mean result of accumulated data and that the sequence is more important than the chronology. Marked changes in sequence may indicate absence of expected tooth germs or the presence of supernumerary teeth. It should be remembered that frequently the early loss of a primary tooth precipitates the precocious eruption of its adult successor.

Tooth Morphology

Upon mirror and explorer examination of the teeth themselves certain salient observations seem important to the writers. The following is not intended as a comprehensive undeviating pattern, but only as a suggested example. Some orderliness is, of course, important, but a "systems" examination usually becomes just that and the important adventure of careful observation is lost.

We like to view the teeth first as individual units with particular attention to morphology. Are the teeth of average size and form? Deeply notched central incisors, because of the restricted development of the central lobe, may be considered pathognomonic of congenital lues, but not all hard, smooth, shovel-shaped teeth are the result of this disease. They may only reflect a hereditary constant. Elevation of the mamelons with well defined notches between the centers of development, accompanied by a highly translucent "blue" line at the incisal edge of the newly erupted adult central incisor, usually describes a well formed tooth. When these teeth appear "flat" and opaque, and

though complete but ill defined centers of development are present, you can generally expect a poor quality dentition with open fissures in the molars and usually a greater susceptibility to decay and abrasion.

"Normal" teeth are not rough and pebbly. When the dentition appears as such, the condition can usually be associated with some other event in the medical history of the child such as resistant rickets. Abnormal pits on the smooth surfaces of the teeth can often be traced back to some prolonged febrile episode such as the exanthematous diseases during the coronal development of the tooth. This "pitting" is not necessarily bilaterally symmetrical. One tooth germ may be in an "active" period of development, while its counterpart in another quadrant of the mouth may be in a "resting" period during the febrile episode. It should be remembered that though the genetic organs of the enamel and dentin which lay down the matrix for these calcified structures enjoy a vascular circulation, there is no demonstrable circulation of the fully developed and calcified tooth.

Occasionally we may see a singular condition such as the *opalescent tooth* which abrades easily. X-ray examination of such teeth may reveal an almost total absence of the dental pulp which characterizes true odontogenesis imperfecta and can be further identified by a history of the condition in multiple antecedents. Such teeth, though poor in character, are not readily susceptible to decay.

Sometimes imperfectly formed enamel leaving areas of very thin or absent "patches" on the crown occurs which leaves the exposed dentin stained a deep brown. Again, poorly formed enamel may manifest itself by appearing volumetrically intact, but of a soft, buttery colored consistency which describes another form of *amelogenesis imperfecta.* Such teeth also have a satisfactory formation of dentin and pulp and are also unexpectedly resistant to dental decay.

Again, on rare occasions, extremely smooth, hard, white, slightly diminutive teeth may be scattered about the dental arches. Upon x-ray examination multiple absences may be revealed. Such teeth are almost invariably associated with other signs which identify a total *ectodermal dysplasia.* On the other hand, the rather frequent absence of such teeth as the superior adult lateral incisors or the inferior second premolars may be encountered without signs of any other ectodermal disorder. Often the absence of these teeth is associated with a familial pattern. On rare occasions a vicarious absence of any of the teeth may occur. Early discovery of this idiopathic pattern by full x-ray examination of the dental arches before age five may save the child orthodontic complications. From this same x-ray examination the early discovery of supernumerary teeth, particularly in the premaxillary and inferior premolar regions, may be revealed. Often it is necessary to surgically

recover these extra teeth at an early stage to prevent serious malocclusion of the normal dentition.

Color and Staining of Teeth

As mentioned in the cases of agenesis of the special tooth structures, color often plays an important role in dental examination. The blue to black teeth witnessed in erythroblastosis fetalis are a good example. This condition is characterized generally by pigmentation in the primary dentition and the definite limit of vital staining to the neonatal line of dental development, but we have seen pigmentation of the occlusal cusp in the first permanent molar in a child who had extreme jaundice after erythroblastosis fetalis. This condition should not be confused with silver nitrate stains sometimes seen in children who have had dental care. Vital staining of the tooth structures may also occur in varying hues of yellow and brown in diseases such as congenital atresia of the bile ducts. Occasionally, vital staining or "mottling" of the teeth may occur because of extra physiological chemical influences on the structure during development. Such is the case of mottling present when excessive amounts of sodium fluoride are endemic to the water supply.

Surface stains of the teeth may be due to fungus residing in the embryonic surface membrane covering the teeth. They may be due to chemical influences such as iron therapy in a suspected anemia and are sometimes thought to be the result of some idiopathic hemolysis, the products being deposited on the teeth via the saliva.

Calculus

Hard calculus is rarely deposited on the teeth of young children, but as they approach adolescence this precipitation process, particularly on the lingual surfaces of the inferior incisors, is a common finding. Sometimes in the presence of metabolic disease such as diabetes there may be observed heavy deposits of calculus on the buccal surfaces of the superior adult first molar as well as the inferior incisors. Such deposits may cause irritation to the gingival mucosa and should be removed.

Dental Caries*

In continuing his examination of the teeth with the mirror and explorer, the doctor should keep in mind the order of vulnerability of decay in susceptible teeth: First, the most likely areas are in the deep grooves and fissures, particularly in the adult dentition. The next areas

* See also page 953.

most likely to be involved are those at the contact point between the surfaces of the teeth. For this reason early x-ray examination by the dentist is important. The next most likely area is at the marginal gingivae, and such lesions are easily identified with the explorer and easily overlooked without this procedure. Finally, the proximal areas of the anterior teeth may become involved. Occurrence in this latter instance invariably identifies the highly susceptible patient, and rapid measures of control should be begun.

Lesions and Infections of Investment Tissues

After observing the size, shape, presence and color of the teeth, attention may be turned to the investment tissues of the teeth. Bony dysplasias such as may occur in renal rickets can only be observed roentgenographically. Bony recession of the alveolar crest except in the presence of skeletal disease is rare. Rare, also, in children is loss of bone due to traumatogenic occlusion of the dentitions. Rarefaction due to dental alveolar abscess is, of course, all too common. By and large, bony defects will be revealed without search during a routine examination because of the acute symptoms. Rare occurrences such as osteogenesis imperfecta will be first identified elsewhere in the skeletal system. It is possible to mistake such diseases as an eosinophilic granuloma for an alveolar abscess; consequently all abnormalities should be investigated by x-ray.

The gingivae, tongue and oral mucosa should, of course, be noted. Changes in color or texture can be readily observed.

Blood dyscrasias such as the leukemias or agranulocytoses are usually identified before oral symptoms become acute. However, the initial diagnosis of purpura is not uncommon from observation of petechial hemorrhages present in the oral mucosa. We have known of instances in which neglect of other symptoms delayed the hematological diagnosis of infectious mononucleosis until oral examination was done.

The signs of drug and chemical intoxication of the marginal gingivae are well known; yet we believe they are more easily identified by the observer who routinely examines these tissues in their normal state. The "blue" line of heavy metal intoxication or the sometimes only slight swelling of the mucosa of patients taking Dilantin are more quickly recognized by the initiated examiner.

Frank infections of the gingivae and oral mucosa, unless they reach the proportions of acute distress, are often neglected by both patients and doctors. The slight swelling and redness of the marginal gingivae can be confusing. Effects of malnutrition and subclinical infections, or both, may present the same picture. At any rate the presence of such signs deserves exhaustive nutritional and clinical laboratory tests to

identify the cause. Scurvy does not always present acute manifestations, and by the same token gingivitis due to poor hygiene or infections deserves detailed investigation.

Viral stomatitis, mycotic infections, virulent cocci infections, so-called Vincent's disease and aphthous stomatitis are usually apparent enough in the acute stages to command attention and need not be discussed here.

ORAL HYGIENE

The instruction for good oral hygiene is the pediatrician's responsibility, and yet we often find that he neglects this opportunity or, worse, minimizes its importance. It is discouraging to find that the presence of swollen cyanotic mucosa seen in congenital heart disease such as tetralogy of Fallot, or the monumental elevations of the gingivae sometimes seen in Dilantin therapy are used as an excuse by the pediatrician to encourage neglect of toothbrushing. These are the very cases in which instruction in good oral hygiene should be prescribed.

The pediatrician has the opportunity to examine the mouth and teeth and point out the importance of good hygiene or evidence of neglect long before the dentist may see the child. Instruction in home care can be relatively simple. Recommending the use of a small but firm brush and almost any dentifrice accepted by the American Dental Association, effort should be made to impress the patient and his parents with the importance of brushing the teeth. Good hygiene unquestionably aids in controlling gingival infection and the initiation of the carious dental lesion. This has been proved over and over again by reliable clinical studies.

Instruction should not be complicated and should begin as soon as the primary teeth are present. A time table such as brushing the teeth as soon as possible after eating and always before retiring should be recommended. If very young children are taught to include toothbrushing every time their face is washed and their hair is combed, it will become a part of the "feeling" of total hygiene and will not be neglected when they are more mature. A "technique" of brushing is to be discouraged in young children—they should simply be admonished to brush inside and outside and on top of their teeth, making sure that they brush next to the gums. Parents should help occasionally, but they should also be warned not to be impatient with childish ineptness and that this intimate act of hygiene should become the responsibility of the child as early as possible.

Though it is regrettable, there is no doubt that it is still true that some dentists, either through ignorance or lack of responsibility, refuse or discourage adequate dental care for children. When the pediatrician

finds this true upon an attempted reference to the "family" dentist, he should without compunction of ethics advise the parents to seek another referral. Most urban areas now have "specialists" known as pedodontists or at least dental practitioners who limit their time in part or in whole to the care of children. Most "good" dental practitioners reserve part of their time for children and only refer the most difficult cases to the pedodontist.

The pediatrician can be of immeasurable help in preparing patients and parents for the child's first visit to the dentist. The age of referral is largely dependent on the pediatrician's examination and judgment of the child's dental needs. He should have in mind that even before all the primary teeth are erupted, some children require care, but that others may not need attention until late in the preschool age. Any doubt as to either disease or development should, of course, prompt a referral.

Most dentists greatly appreciate the physician's opinions of the physical and psychological condition of the child. The dentist and the parent have a better chance of understanding each other if they are both "briefed" by the pediatrician about the problems of a sick or "difficult" child. His judgment as to the length of appointments, use of anesthesia, special approach, even the time of day for appointments for the child with special problems may mean the difference between achievement or failure with the dentist.

This paper does not lend itself to extensive discussion and classification of patient management during dental care, but, certainly, the "problem" child deserves dental care as much as the "normal" child. It must be remembered that even routine dental service often demands more cooperation of the child than any other health service offered him.

Where chair service is impractical hospitalization and the use of general anesthesia may be resorted to. This service is available in most urban areas. Many spastic, incompetent or even young "difficult" children requiring extensive interference are better served in this manner. This is invariably a "team" job, and the advice of the pediatrician along with a thorough physical work-up, including pertinent clinical-laboratory data, is paramount. Even when an "office" procedure under general anesthesia is prescribed, an examination by the pediatrician immediately preoperatively is mandatory.

SUMMARY

In summary, attention has been called to the fact that the thorough examination of the child's mouth is an integral and important part of a medical assay of the total health and growth and development of the child. The findings from a careful oral examination of the child are

almost legion, but only a few have been discussed. The methodology used by dentists for such examination can at least in part be applied successfully by physicians interested in child health. The technique is relatively simple and not unduly time consuming. With a slightly increased armamentarium the pediatrician can become familiar with both the normal and pathological dental and oral conditions and work more effectively with the dentist for the welfare of the child's over-all health.

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203 Commonwealth Avenue
Boston 16, Massachusetts (Dr. Sisson)

CARIES CONTROL IN PEDIATRIC PRACTICE

MAURY MASSLER, D.D.S.

More than 90 per cent of the grade school children and approximately 50 per cent of the preschool children between three and six years of age, visiting the pediatrician for preventive care, have one or more carious teeth. Most of these teeth are unfilled. Many mothers are unaware of the tooth decay, and will remain unaware of it unless the pediatrician calls it to their attention. Parents often do not seek dental care for children until a toothache develops. Parents are usually quite surprised when the decay reaches the pulp, causing extreme pain and infection of the jaw. This is hardly a good introduction of the child to the dentist, and certainly not in keeping with sound principles of preventive medicine.

Prevention of disease is the primary goal of the pediatrician. He carefully examines the throat, the tonsils and the tongue at every visit even when there are no complaints. But many pediatricians feel that dental disease is not within their province and leave the examination and care of the teeth to the dentist.

This clinic will attempt to clarify the role of the pediatrician in the control of dental caries and indicate how he can help the pedodontist implement a successful program of caries prevention.

PEDIATRIC TEETH EXAMINATION

The following steps applied to daily pediatric practice would do more to help prevent dental decay and tooth loss than all the dentifrices now sold to the American public. The technique is neither difficult nor time consuming.

1. Examine the teeth of *every* child coming to the office. Do this just before looking at the throat and tonsils. The average time required

From the Department of Pedodontics, University of Illinois College of Dentistry, Chicago.

is 50 seconds. The equipment is a tongue blade and intraoral light. Place the child in a comfortable position and proceed as for a throat examination.

Begin with the *lower left last molar* and proceed around the lower arch to the right side. Touch each tooth with the tip of the tongue blade. It is well to count aloud as you proceed to gain the child's cooperation and to allay his fears. Count from one to 10 for the preschool child and from one to 12 for the grade school child. (There are 10 primary or deciduous teeth in each arch from ages two and one half to six. The first permanent molars appear behind the last deciduous molars at age six.) Repeat with the upper arch, going from the child's left to right.

Inspect the molar teeth again with special care, since dental caries always begins in the occlusal pits and fissures of these teeth.

2. If the child has no cavities, a referral should be made to a pedodontist or dentist with an interest in caring for children for early introduction and a program of preventive dental care. A notation of the name of the dentist should be entered in the record.

3. If the child has one or two small cavities, insist that he be taken to a dentist at once for fillings. Check for fillings at the next visit.

4. If the child shows numerous cavities, institute caries control measures and refer to a competent dentist at once.

The most effective method of arresting active dental decay (at present) consists in minimizing the *excessive* intake of sugars *between* meals. After reviewing the diet, suggestions for substituting cold cuts, cheeses, fruits and other more healthful foods for the cariogenic confections consumed in large quantities by many American children between meals are indicated.

The pediatrician should guide the dietary habits of his preschool and grade school children with the same care that he shows the infant. When the child is healthy and vigorous and growing well, the tendency is to omit dietary supervision. But when dental disease becomes evident, the pediatrician has the responsibility to review the diet with a view towards better systemic as well as dental health.

DENTAL CARIES

In order to bring the problem of dental caries into better focus, it would be appropriate to review briefly the major characteristics of dental caries, since this disease too often receives only superficial attention in most medical school curricula. Once the main features of the disease are understood, it is not difficult to apply the various methods of caries control.

The Caries Process

Dental caries is the result of bacterial attack of the hard substance of the tooth, beginning on the surface and progressing inward towards the pulp and resulting in the formation of a cavity. The primary attack on the enamel or dentin is made by acid metabolites produced by acidogenic organisms (lactobacilli, streptococci and others) living on a fermentable carbohydrate substrate on the surface of the tooth. Acid decalcification is followed by proteolysis of the organic matter with the result that dental caries is a slowly progressive and intermittent process.

Incidence

Dental caries is primarily a disease of childhood. It begins early in childhood as soon as the molar teeth are erupted and accumulates rapidly, with acute exacerbations between four and eight years and 12 and 16 years of age. The rate decreases substantially after the age of 30 years.

Dietary Factors

The most important contributory factors in the initiation and progression of dental decay are the readily fermentable carbohydrates (particularly the sugars) which adhere to the surface of the teeth.[2] These can be metabolized by the acidogenic organisms within a few minutes. The pH on the tooth surface (as measured by an antimony electrode) will drop from 7.0 to 5.4 within two minutes after a single glucose or sucrose rinse.[6] Effective caries control depends on preventing the accumulation of fermentable sugars on the tooth surfaces. This can be accomplished by (*a*) restricting the sugar intake to mealtimes when the natural cleansing action of detergent foods and saliva prevents the accumulation of sugars on the tooth surface or (*b*) by brushing the teeth immediately after sweets have been ingested.

Other dietary or nutritional factors do not appear to play any significant role in the production or prevention of human caries. In this respect it should be pointed out that the role of calcium and vitamin D in caries prevention is practically nil.

Genetic Factors

There is reasonably good evidence from both animal and human studies to suggest that genetic factors may also influence susceptibility to decay. Approximately 5 to 7 per cent of the population are caries-immune in spite of dietary indiscretions and poor oral hygiene. Caries immunity can often be traced through family groups. The mechanism

of this immunity is not known. It may lie in a potent bacteriostatic property of the saliva.

Extreme susceptibility to dental decay (rampant caries) afflicts approximately 7 per cent of the population. These persons suffer severe attacks of decay even when they practice good oral hygiene and sugar restriction. In them, dietary indiscretions and poor oral hygiene are catastrophic and result in a complete destruction of all the teeth in the mouth so that they become edentulous before the age of 25 years. Rampant caries seems to follow a definite familial pattern and is probably the result of an inherent susceptibility to dental decay aggravated by excessive consumption of sugars and poor oral hygiene.

Pattern of Caries Attack

Dental caries begins in the pits and fissures on the occlusal (biting) surfaces of the molar teeth. These are the most susceptible areas, and the pediatrician should always examine the occlusal surfaces of the posterior teeth for signs of decay. The second site for caries attack are the proximal contact surfaces between the molar teeth. These are difficult to examine, even by the dentist—who usually obtains intraoral roentgenograms to make his inspection more complete. The third (and least frequent) site for caries attack are the necks of the teeth (cervical areas) near the gingivae.

Dental caries spreads from the molar teeth anteriorly. As long as only the posterior teeth are affected, the disease may be considered mild. When the bicuspid teeth become involved, the disease is considered to be moderate. When decay spreads to the approximal surfaces between the upper anterior teeth, it is considered severe and the child rates as being caries-susceptible. If the lower anterior teeth become involved, the condition is called "rampant caries." By this time, too, the cervical areas of the molar, bicuspid and sometimes also the incisor teeth are attacked by caries.

Caries Control

Caries control (i.e., limitation of the spread of the lesions, once the lesion has appeared) may be achieved by (*a*) early and regular visits to the dentist and (*b*) elimination of fermentable sugars between meals.

Caries Prevention

In the average child a significant degree of caries *prevention* (preventing the appearance of new lesions) can be achieved by the following: (1) elimination of fermentable sugars between meals, (2) tooth-

brushing immediately after each meal, (3) topical application of fluorides to the enamel as soon as the teeth erupt, (4) the addition of one part per million of fluorides to the communal drinking water supply.

The pediatrician can do a great deal towards implementing a program of caries control in his patients by insisting that children under his care visit the dentist regularly and that they begin to do so *before* the teeth show gross evidence of decay. The pediatrician can also do a great deal to help the pedodontist to arrest the decay, once it becomes apparent, by introducing the parent and the child to the concept of caries control by sugar control. The pediatrician is regarded as the authority in matters of the child's health. He can encourage the mother and child to cooperate with the pedodontists' program or nullify these efforts by a too casual attitude concerning dental health.

Caries prevention and caries control measures are especially indicated for children who would suffer excessive hazards from tooth extraction (the child with rheumatic heart disease, kidney disease or hemophilia).

CARIES CONTROL VIA SUGAR CONTROL

An extensive scientific literature has accumulated during the past two decades which proves beyond reasonable doubt that the consumption of excessive amounts of refined sugars, particularly in the form of rapidly fermentable carbohydrates (especially sugars) which stick tenaciously to tooth surfaces, leads to a marked increase in the caries attack rate. Thousands of caries-active children and adults have had the disease completely arrested by a drastic reduction in sugar intake, i.e., *the elimination of sugars which are consumed in excess of nutritional needs.*

During the war these clinical observations were duplicated in almost an experimental fashion in large groups living in Europe. As the annual consumption of sugars decreased during the war in The Netherlands, Norway, Sweden and England, the average caries attack rate fell in proportion. After the war, when sugar in the form of candies, cookies and the like again became freely available, the incidence of caries rose again, in proportion.[7]

Other Effects of Excessive Sugar Intake

Excessive sugar intake between meals (above nutritional needs) constitutes a hazard not only to the teeth. Excessive consumption of between-meal sugars is an important factor in the loss of appetite at mealtimes for essential foodstuffs such as meats and vegetables. The common complaint by parents that their children have no appetite and fail to eat at mealtimes may often be traced to the enormous consumption of refined sugars between meals. In this way excessive sugar con-

sumption predisposes to poor nutritional balance, since it literally pushes out from the dietary the other necessary proteins, minerals and fats.

The consumption of sweets in excessive amounts during early childhood may be a reflection of a psychologic problem which causes the child to seek gratification from eating because his needs are not being met in other ways. Thus the sugar appetite in the child and the alcohol appetite in the adult have many points in common from both the metabolic and psychologic viewpoints. Small amounts of sugar or alcohol may stimulate the appetite and lead to obesity. Excessive amounts lead to malnutrition by depressing the appetite. It is interesting to note that rampant caries has its highest incidence in the thin, undernourished child who subsists largely on between-meal snacks or sweets. The obese child tends to have a lower than average caries attack rate.[3]

Snack Habits and Dental Caries

If a person were to consume sugars only at mealtimes, the caries attacks would be minimized by the buffering and detergent action of the saliva and alkaline foodstuffs. However, when refined sugars are ingested *between meals and at frequent intervals,* the teeth are subjected to an almost continuous series of assaults by the acids formed on the tooth surfaces. The caries attack rate thus becomes enormously increased by the habitual consumption of sugars between meals. It is not so much the total quantity of sugars consumed during the day that is important as the *number of times* a sugar meal was taken. An occasional "candy binge" is not as injurious as the habitual daily consumption of caramels or chewing gum.

Correcting the Diet

Caries control via sugar control should be initiated by the pediatrician as early as possible. Correcting the diet and eliminating excessive amounts of between-meal sugars are not difficult if the physician will first obtain a careful history of the daily intake of the child for a period of approximately one week. Careful review of the diet often reveals peculiarities in eating habits.

Individual caries susceptibility determines to a large extent the amount of fermentable carbohydrate which is cariogenic. A very susceptible child may suffer great destruction of the teeth from what appear to be only "normal" amounts of sugars. Other children may consume large amounts of candies without severe damage. The degree of caries susceptibility of the child must be pointed out to the parents

when they object to suggestions that the destruction of the child's teeth may be due to overindulgence in sweets.

Many parents find it difficult to limit between-meal snacks and sweets to their children. A more effective technique is to offer an apple or potato chips instead of candies or cookies or a sandwich containing meat or cheese instead of one containing jams or jellies. A healthy child will enjoy an apple with as much gusto as a piece of cake, and will accept noncariogenic popcorn or potato chips instead of caramels with no loss in caloric intake. A lunch consisting of a meat-sandwich and milk is just as satisfying as and far more healthful than one made up of a sweet-roll and soda-pop. In fact, if given a free choice, most young children will select the fruit and the sandwich over the cake and caramel as a steady diet. Only those with already conditioned appetites prefer the sweets.

If parents are considerable consumers of candies, cookies and chewing gum, one cannot expect the child to deny himself these confections. It is important that the physician recognize the impact of family food habits upon the child. Often parents will make a sincere effort to change their own dietary habits in the interests of their children's health.

It would be pertinent here to point out that the appetite for sweets is acquired early. The pediatrician can do his part in discouraging the excessive consumption of candies by offering his patients a toy balloon or trinket as a reward instead of a lollipop.

CALCIUM, VITAMIN D AND CARIES

Vitamin D and calcium compounds have often been invoked as therapeutic agents for the control and even prevention of dental decay. Thousands of calcium tablets, vitamin D pills and gallons of milk have been consumed by children and pregnant females toward this end. However, the past two decades have indicated clearly that such therapeutic measures are not effective, since the caries attack rate has increased steadily during that period. There is certainly no rational basis, at present, for the continuation of these empiric and ineffective therapeutic measures. This does not in any sense negate the value of calcium and vitamin D in the growth and calcification of bone. However, they seem to have no value in caries prevention.

FLUORIDES AND CARIES

Fluorides in Water

It has been demonstrated that the caries attack rate is much less in areas where the fluoride content of the water supply is one part per

million or more than in nonfluoride areas. It has therefore been proposed that fluorides be added to communal water supplies to reduce the incidence of caries. This hypothesis has been subjected to careful and intensive testing over a 10 year period in Newburgh and Kingston, New York; Grand Rapids, Michigan; Evanston, Illinois; and other cities. The inverse relation between water-borne fluorides and dental caries is now one of the most thoroughly tested and authenticated relationships in all modern medicine. These studies have demonstrated conclusively that the addition of minute amounts of fluorides (one to two parts per million) regularly reduces the caries attack rate approximately 40 to 60 per cent with perfect safety and absolutely no systemic effects and at a low cost (approximately eight to ten cents per person per year).

Inevitably there have been objections to this procedure by small but vocal groups. Some object to the fluoridation of water as "socialized medicine" or "mass medication," forgetting that the chlorination of water, the pasteurization of milk and the addition of iodides to table salt are similar preventive measures. The addition of fluorides to the water is not a therapeutic procedure (it does not heal the carious lesion); it is a *preventive* measure.

Other groups object to the fluoridation of water on religious grounds. Still others object on the basis that fluorides are protoplasmic poisons. In powder form or as strong solution, fluorides are poisons and have been used as insecticides. In a concentration of one to two parts per million fluorides are not at all poisonous. Chlorine is also poisonous when concentrated as a gas, but not when used in weak dilutions to purify the water.

The objections offered by small groups against the addition of fluorides to drinking water are primarily emotional and not based on any real evidence. These groups will unfortunately delay the fluoridation of the drinking water in some communities, but will not, in the long run, stop progress in the mass reduction of caries by water fluoridation, because the scientific evidence to support the use of water fluoridation as a public health measure for the reduction of dental caries is now overwhelming.

Fluorides in Foods

Many foods (fish, tea, many vegetables) contain relatively large amounts of fluorides (40 to 100 parts per million). These fluorides have no effect in reducing the incidence of caries probably because the fluorides in foods are in insoluble combinations and not available for action on the teeth. Apparently fluorides must be in water solution and in a soluble, ionic form to act in caries reduction.

Fluoride Tablets

Though there is excellent evidence to show that the addition of fluorides to the communal water supply is effective in reducing the prevalence of dental caries, there is no evidence to support the suggestion that fluorides in the form of tablets or lozenges would be similarly effective. Commercial preparations which contain calcium fluorides plus multiple vitamins have nonetheless been offered for the control of dental caries. There have been no acceptable studies made to support such claims. Such tablets contain insoluble calcium fluorides which are not absorbed from the intestinal tract and are therefore nontoxic. They are also inactive against caries.

Topical Applications of Fluorides

The relation between fluorides in the water supply and reduced caries experience in children has led directly to experimental studies on the effect of topical applications of fluorides to the teeth. Knutson and Armstrong (1942), Cheyne (1942), Bibby (1944) and many others have demonstrated conclusively that the topical application of fluorides to the teeth of children is an effective method of preventing the initiation of new enamel caries. Solutions of sodium fluoride 2 per cent were applied only to the teeth on one side of the mouth. The untreated side in the same child served as the control. This procedure showed a reduction of 25 to 40 per cent in the incidence of new caries in the treated side over the untreated side after one to three years of observation.

The topical application of fluorides has none of the objections offered to its systemic administration, since the material is not swallowed and is therefore not toxic. The accepted method of sodium fluoride applications consists in an initial, thorough dental prophylaxis followed by a minimum of four treatments within a relatively short period (two to three weeks).

Fluorides are effective when applied to the recently erupted teeth of children, but not in adults. Newly erupted enamel is receptive to the fluoride ion, the apatite molecule changing from the soluble hydroxyapatite to the less soluble fluor-apatite. The adult enamel surface is impregnated over a period of years by ions derived from the saliva and is no longer receptive to the fluoride ion.

Topical applications of fluoride solutions to the teeth of children are *not* contraindicated in areas where fluorides have been added to the drinking water. The added fluorides are not ingested and will not cause mottling, but may further increase the resistance of the enamel surface to acid etching.

Fluoride Dentifrices

On the basis of the efficacy of topical applications of sodium fluoride solutions to the surface of the newly erupted enamel in reducing the incidence of new lesions, it was logical to assume that sodium fluoride might be effective if added to dentifrices or mouthwashes. Unfortunately repeated trials have shown that sodium fluoride in the form of a dentifrice or mouthwash is not effective in reducing dental caries (Bibby et al., 1946). The fluoride ion probably combines with the calcium or other metallic ions present in the dentifrice and becomes unavailable to the enamel.

Recently, Muhler et al.[5] demonstrated that stannous fluoride is more effective than sodium fluoride solutions when applied to the enamel of hamsters' teeth or human teeth. He showed also that when stannous fluoride is added to a noncalcium dentifrice, it is effective in protecting the enamel against attack by caries. Although a vast advertising campaign has already sold millions of dentifrices containing stannous fluoride, it is still too early to say with certainty that these are effective in reducing dental decay. Muhler's results have not yet been duplicated by other investigators.

ORAL HYGIENE AND CARIES

There is no doubt that brushing the teeth immediately after each meal would significantly reduce the incidence of dental decay, probably by reason of removing the carbohydrate substrate before bacterial action produces significant amounts of acids. Fosdick[1] showed that regular brushing of the teeth immediately after meals reduced the caries attack rate by as much as 40 per cent. The amount of toothbrushing and its timing are far more important than the material contained in the dentifrices.

Unfortunately toothbrushing immedately after each meal is not practical in modern society. In fact, it is rare to discover any person who brushes his teeth at other times than on arising or before retiring. Toothbrushing performed at the beginning or end of the day serves a cosmetic function, but has no effect in preventing dental caries because it does not remove the acids on the tooth surfaces at the time they are formed.

Nonetheless, the search continues for a magic toothpaste which will prevent dental caries. Ammonium, chlorophyll, penicillin and a variety of antienzymes have been recently added to dentifrices with many promises by the manufacturers that these would halt all decay. This has sold millions of dentifrices, but has not yet significantly reduced the incidence of dental decay. It is generally agreed that the main in-

gredient in good oral hygiene is the toothbrush and the "elbow-grease" used rather than the dentifrice.

EARLY DENTAL CARE

Removal of the decay by the dentist and filling the cavity are still the most certain methods of arresting the progress of the lesion. Unfortunately, few patients visit the dentist early enough or regularly enough so that early lesions can be detected and filled. Towards this end the pedodontist must have the active cooperation of the pediatrician.

Somewhat less than 20 per cent of all children requiring dental treatment receive it at an early age. In all too many instances the child's first visit is made only after there has been pulpal infection, with periapical pain. This is hardly a good introduction to dentistry. The child's first experience with the dentist conditions his attitude towards dentistry and dental procedures. In general, the three year old is ready to make the acquaintance of the dentist. If the first experience is a pleasant one, and nonpainful, the child will be better prepared to accept dental procedures when they become necessary. Most dentists complain that they do not see the child early enough and are therefore confronted with emergency problems initially rather than a healthy cooperative child. The pediatrician can do a great deal to help overcome the tremendous loss of teeth caused by dental caries if he would refer his young patients to the dentist *before* severe pain occurs.

The primary dentition is completed between two and two and one-half years of age. In susceptible children dental decay usually begins in the molar teeth before the age of three, and even in the average child at least four carious lesions can be expected before the age of six. It is therefore suggested that the pediatrician refer the patient to a dentist for examination and careful inspection of early lesions at approximately two and one-half to three years of age. When cavitation is observed, the pediatrician should insist upon *immediate* treatment by a pedodontist, lest extension of infection through the pulp and into the periapical bone result in loss of the tooth.

Referral to the Dentist

The admonition to "Go see your dentist" does not constitute a referral. All too many do not go to the dentist. Some encounter dentists who do not care to treat children. It is not uncommon for pediatricians to complain that they have sent patients to a dentist only to find that the dentist would not or could not take care of the child. This is frustrating. The pediatrician would do well, therefore, to discover and

become acquainted with the local dentists and/or pedodontists who are able and willing to treat children. The referral should be definite and as precise as the referral to an otolaryngologist. Perhaps the simplest approach is first to obtain a list of names (from the local dental society or local American Society of Dentistry for Children) of practitioners who are accustomed to treat child patients.

A further responsibility of the pediatrician lies in finding a dentist who is able to care for the handicapped child. In those cases cooperation with the pedodontist in providing a bed at the hospital and a competent anesthesiologist would extend dental services to many more handicapped children than now receive them.

SUMMARY AND CONCLUSIONS

The responsibility of treating and repairing the teeth rests with the pedodontist. However, the pediatrician can do a great deal to promote and implement a program of preventive dental care and caries control in the preschool and grade school children.

The pediatrician should insist that the child see the dentist some time before his third birthday, *before* extensive dental repair becomes necessary. It is not wise to wait until decay becomes clinically evident any more than one waits for disease before instituting a preventive program of immunization. The pedodontist would like to see the child early in order (*a*) to institute preventive measures by dietary control of sugars, the application of topical fluorides and teaching good oral hygiene, and (*b*) to minimize the development of fear of the dentist. There is nothing more frustrating to the dentist than to be presented with 10 deep and painful cavities in a child who is extremely fearful of the dental procedures and therefore uncooperative. The greatest cause for dental neglect is fear. This can be prevented primarily by early introduction of the child to the dentist *before* he needs painful treatment.

The pediatrician can do a great deal to reduce the caries attack rate in the American child by discouraging the habitual consumption of cariogenic sugars above the nutritional needs of the child. Dental caries is primarily a bacterial attack upon the hard tissues of the tooth by acidogenic organisms. These organisms produce acid metabolites on the surface of the teeth, using fermentable carbohydrates (sugars) as the substrate. Adherent foodstuffs containing sugar which remain on the tooth surface for more than 20 minutes lead to a marked production of acids on the tooth surface with enamel decalcification and initiation of the caries lesion. The American child leads the world in the consumption of sweets and in dental caries.

Good dietary habits like hygienic habits must begin early in child-

hood. Supervision of the child's dietary should not cease at infancy. The child's diet should be the joint responsibility of both the pediatrician and the pedodontist.

The fluoridation of water has been demonstrated to safely reduce the caries attack rate. Increasing acceptance of this public health practice by communities is destined to enhance caries control.

Dental health is part of the total health picture. No child can be considered perfectly well if he is suffering from extensive and destructive dental decay. Dental health is therefore a part of pediatric care.

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808 South Wood Street
Chicago 12, Illinois

THE ORTHODONTIST AND THE PEDIATRICIAN

MILTON B. ENGEL, D.D.S.

The congruence of interest of the orthodontist and the pediatrician lies in their concern with certain general and specialized aspects of growth and development in the child. The normal development of occlusion hinges on the orderly emergence of the growth pattern of the face and jaws. This, in turn, must be correlated with somatic growth.

NORMAL OCCLUSION AND THE TYPES OF MALOCCLUSION

The criteria for normal occlusion were not clearly stated until the latter part of the last century when Kingsley,[20] Case[10] and Angle[1] published their views. Angle emphasized that there was a well defined and constant dorsal-ventro relation between the maxillary and mandibular molars in the normal dentition, and upon this he based his classification of malocclusion. In brief, he designated three groups or classes of malocclusions which are illustrated by Figure 39. These are:

Class I, in which the molar relations of the maxilla and mandible are correct, but other irregularities such as crowding are present.

Class II, in which the mandible appears to be retruded or the maxillary teeth appear to be prominent.

Class III, in which the mandible appears prominent or protrusive. In both class II and class III malocclusions the molar relations depart from the normal.

This classification of malocclusion has been in use for the past 50 years and has been valuable for diagnostic standardization and for

From the Department of Orthodontics, University of Illinois, College of Dentistry, Chicago.

purposes of communication. In recent years, however, greater emphasis is being placed on the spatial relations of the dental apparatus to the entire craniofacial complex. Ultimately, it appears likely that the classi-

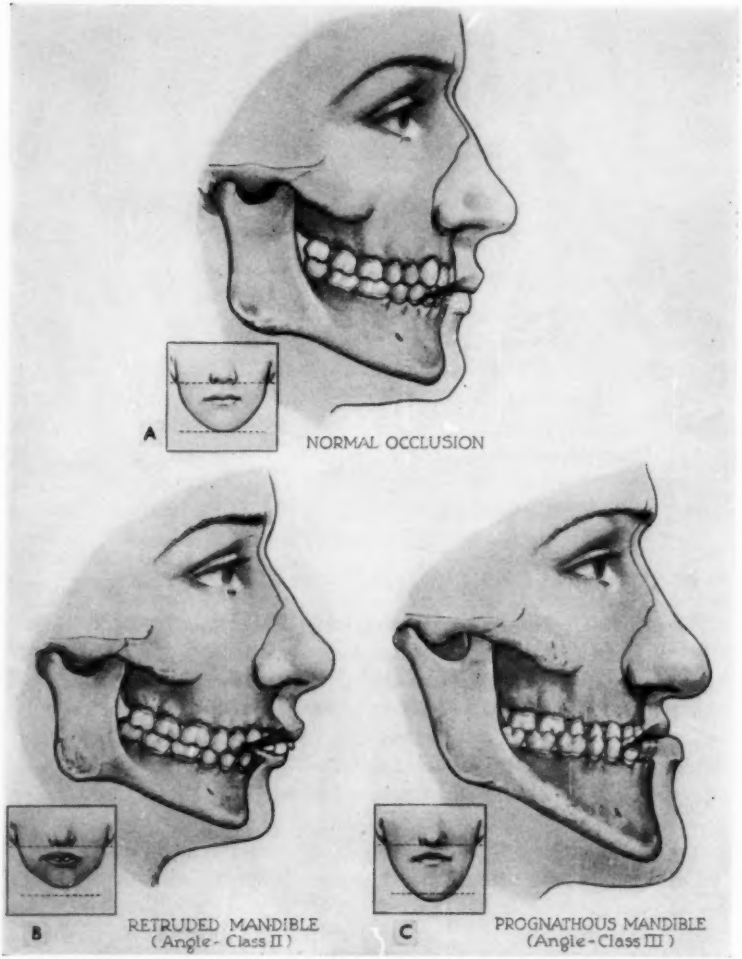

Fig. 39. Illustrating the relations of the maxillary and mandibular teeth in normal occlusion and in malocclusion. (From The Atlas of the Mouth, courtesy Drs. M. Massler and I. Schour.)

fication or diagnosis of malocclusions on the basis of interdental relations alone will give way to a diagnosis based on more fundamental skeletal features.

SOMATIC GROWTH, CRANIOFACIAL GROWTH, AND THE DEVELOPMENT OF OCCLUSION

The development of dental occlusion is closely allied with the growth and development of the skeletal structures which support the teeth. The early growth studies on the head and face were those of Hellman[17] and of Goldstein.[15] They were based on data compiled from measurements of large numbers of children at varying ages. These were significant contributions, but because they were cross-sectional studies some misconceptions concerning the general nature of the changes arose. On the whole one had the impression that the process of growth in the head and jaws was almost sporadic and discontinuous. It is a general defect of cross-sectional studies that significant individual variations with their peculiar time-linked characteristics may either cancel out or give spurious peaks to a growth curve.

Until recently longitudinal studies have been rare. The classic example, pertinent to this day, is the one recorded by Buffon in 1777 on the statural growth of a French nobleman's child.[27] During the past 25 years more attention has been given to the serial study of individual children. Orthodontics owes much to T. W. Todd and his Western Reserve group for the impetus they gave to this revealing approach.

With the development in 1931 of a headholder and of standardized x-ray technique simultaneously by Broadbent of Western Reserve[4] and Hofrath in Germany[18] it became possible to take accurate oriented lateral and postero-anterior films of the same child over the entire period of its growth. An early report dealing in a qualitative way with the growth changes in the skull was made in 1937 by Broadbent.[5] In 1941 a more detailed description was published by Brodie,[6] who studied children from the Western Reserve series from three months to eight years. From the work of Broadbent and Brodie it has been demonstrated that each person has an orderly pattern of craniofacial growth which emerges in time. The facial structures continue their growth after the cranial parts have decelerated. This accounts for the apparent emergence of the face from under the cranium as the child grows up. It is notable that within the face there are no sharp inflections in the relative rates of growth, and thus, in a general way, the proportionality of the parts remains fairly constant. These generalizations are illustrated in Figures 40 and 41, which were made from tracings of the roentgenograms of one child over a period of 17 years. In any heterogeneous group such as found in the child population of the United States the possibilities for variation in the facial pattern within the bounds of "normality" remain great.

More recently an attempt has been made by Nanda[23] of the Child Research Council of the University of Colorado to relate growth rates in the head with general body growth. It appears that the prepuberal

Fig. 40.

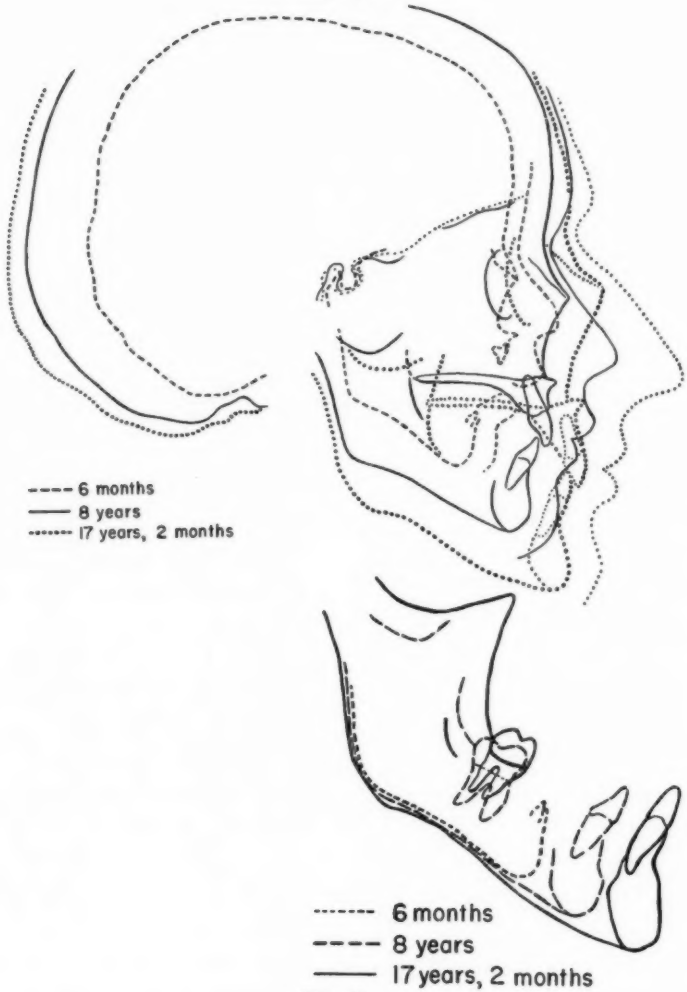

Fig. 41.

Fig. 40. The orderly emergence of the growth pattern of the craniofacial skeleton over a 17 year period is shown in the superimposed tracings of the roentgenograms of a male child. (Courtesy of Dr. Allan Brodie.)

Fig. 41. Superimposed tracings of mandibles from Figure 40.

acceleration or "spurt" in growth as revealed by statural changes coincides remarkably well with similar changes in the face. Nanda's evidence suggests that the facial growth spurt extends for a somewhat longer period. Sexual differences in the initiation of the acceleration in general body growth also appear to be duplicated in growth of the facial structures. Much remains to be worked out concerning the correlations of general skeletal and somatic growth with growth of the craniofacial area. This is a problem with the most important clinical implications for the orthodontist, particularly in the matter of treatment planning and in prognosis.

The method of cephalometric roentgenology can be a valuable diagnostic aid in the evaluation of the child patient. Single films are useful for undistorted delineation of the craniofacial skeleton and the contiguous soft structure of the face, mouth and pharynx. Serial films are used to reveal the growth pattern of a child or to indicate growth arrests or recovery. This method has been a great aid in evaluating surgical procedures involving the face and jaws and in determining the effects of orthodontic treatment.[7]

The growth of the bones of the jaws is a continuous process, but the eruption of the teeth displays discontinuities and gives the appearance of being considerably less orderly. But in a general way the eruption of the teeth is correlated with development and maturation, and this may be especially true in the transition from deciduous to permanent dentition. For routine office use tooth eruption standards as determined by Schour and Massler,[24] by Hurme[19] and by Meredith[22] are of value. Slow eruption patterns may reflect slow, but normal, somatic and skeletal maturation. In more unusual instances marked delay is characteristic of such frank defects as hypothyroidism, pituitary dwarfism and cleidocranial dysostosis.

Serial studies have been made also on the growth in size of the dental arches and on the development of the occlusion.[25, 26] Generally, when the bite relations are originally abnormal, they remain so. When the arch is crowded in the deciduous dentition, there is likely to be a similar problem in the transitional and permanent dentition. I would not wish this to be construed as a dogmatic statement, since the records of Sillman[25, 26] show that within each person's pattern there lies a considerable latitude of adjustment.

ETIOLOGY OF MALOCCLUSION

A malocclusion is usually the reflection of some fundamental discrepancy in jaw relations or of tooth and jaw disharmony. The early view placed much of the fault on environmental factors, but this concept of etiology must now be considered somewhat naive. More

careful studies, especially the more recent serial analyses, indicate that the pattern of occlusion is present early—probably at least at birth. There are strong indications that genetic factors are of great importance in establishing the pattern of malocclusion.[3] A few of the general and local influences will be considered.

Some severe growth aberrations involving the face appear to have their inception in the fetal period. Though genes may be involved, it is at least as likely that modifications of the internal milieu at critical developmental periods may be responsible for the defects. Warkany's nutritional deficiency experiments leading to cleft palate defects in rats appear to support such a view.[30]

One of the most critical areas for the growth and function of the jaws is that of the mandibular condyle. The microscopic anatomy of the condyle is analogous to that of the epiphysis of a long bone with proliferating cartilage and an ossifying spongiosa, and its role in mandibular growth is correspondingly determinant. Injuries to this center through trauma, infection or metabolic disease may cause a serious dysplasia.[13] In some instances of congenital micrognathia we may be dealing with such a prenatal injury. During the postnatal period injuries to the mandibular condyle can severely affect growth of the mandible, leading to a retruded jaw typical to some class II malocclusions. These deformities were less rare before the advent of antibiotics which abort the spread of middle ear infections to the mastoid and temporomandibular areas. The treatment of lymphoid masses in the pharyngeal and posterior oral areas with radiation can also lead to condylar injury. Traumatic fractures of the condyle do occasionally occur, and this area should be carefully examined whenever a child suffers a hard blow or fall injuring the side of the face or the chin.

In rheumatoid arthritis of children the temporomandibular joint is more susceptible to injury than is generally known. If the disease persists, the growth center may be injured and in consequence the growth of the lower face is severely retarded.[14] The deformity tends to be symmetrical, and the mandible is retruded, giving rise to the "Vogelgesicht" (Fig. 42). A severe class II malocclusion develops, and the bite may be open in the forepart of the mouth. The functional range of the joint is sometimes impaired, and in laminagraphic roentgenography a flattening or erosion of temporal and mandibular parts may be visualized (Fig. 43).

Mandibular prognathism, on the other hand, can be induced by overactivity of the condylar growth center. Most cases of prognathism have their inception from birth or the prenatal period, though a small group arises during the course of the later development of the face. In the parapuberal period, particularly, there may be accelerated mandibular growth, perhaps due to hormonal influences on the carti-

lagenous condyle. In these children the occlusion of the teeth is likely to change as the mandible becomes prominent.

Skeletal growth is affected by such general metabolic influences as nutrition, illness, endocrine activity. As a specialized part of the skeletal system the jaws may also be affected.[28, 29] In protracted and severe

Fig. 42. Profile photograph and lateral head plate of a 16 year old girl who has been ill with rheumatoid arthritis over a period of about 10 years. The pattern of mandibular growth is particularly affected because of involvement of the growth center in the condyle.

illness there may be inadequate jaw growth and the immutable, predetermined tooth mass cannot be accommodated. Malocclusion may also be one of the consequences of certain states of depressed endocrine activity.[11, 12]

There is a large group of malocclusions whose origins are due to purely local factors. Some in this group owe their origin to the premature loss of deciduous teeth. Sometimes when space-maintaining devices

are not used the adjacent teeth will drift because of growth tendencies and biting forces; then the permanent successor is crowded out of its normal position. Tooth relations in the affected dental arch as well as in occlusion may be disturbed.

Many malocclusions arise because of discrepancies in the size of the teeth and of the supporting jaw structures. This type of defect may have its roots in the genetic constitution of the child. In a smaller group the disproportion is due to a combination of an immutable predetermined tooth mass with a skeletal structure which is responsive to the warping influence of an unfavorable muscle environment.

Since the supporting bone of the teeth is quite plastic, it responds readily to pressure from the tongue and circumoral musculature. Compulsive habits involving the lips or tongue will exert significant influ-

Fig. 43. Laminagraph of the temporomandibular joint of a 13 year old child with rheumatoid arthritis showing the flattening and erosion of the articular surfaces of the joint.

ences on the position of the teeth. In this way also thumb sucking will lead to a local distortion of the bone. The upper incisors may be forced forward (labially), leaving a gap in the vertical and horizontal directions between the upper and lower teeth (Fig. 44). The effects of this habit are likely to be less prominent if it is discontinued before the permanent incisors erupt. Thumb sucking is sometimes associated with obesity and other psychological manifestations of oral fixation. Some orthodontists feel that the detrimental effects of the habit on the dentition justify the placement of deterrent appliances or restraints. In my opinion the dental effects of the habit are less serious than the potential consequences of active repression. In any event, since thumb sucking which persists is generally a symptom of emotional disturbance, it is best treated by psychiatric management. An excellent report dealing especially with the problem of the psychic effects of interference by an appliance has been made by Korner and Reider.[21]

ORTHODONTIC TREATMENT

Objectives of Treatment

Parents who seek orthodontic treatment for their children are often most keenly concerned about the cosmetic defect. This is certainly a valid reason for treatment, but the primary objective of the orthodontist is to establish the normal function of the dental apparatus, thus permitting normal mastication, speech and breathing. Treatment also has its prophylactic aspects and is designed to (1) prevent periodontal disturbances created by abnormal stresses on the teeth and their supporting structures; (2) minimize dental caries, to which crowded,

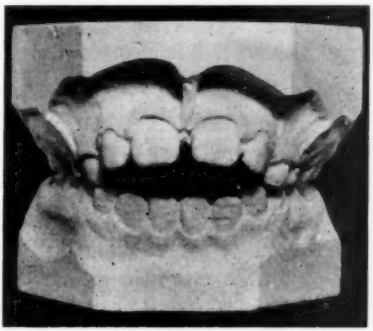

Fig. 44. Frontal view of casts of the dentition of a child who has a thumb-sucking habit. The upper incisor teeth have been forced forward and the bite is open. (Courtesy of Dr. Elsie Gerlach.)

malaligned teeth may be disposed; (3) prevent disturbances in the temporomandibular joint which might be induced by shifts in the position of the mandible caused by the interferences of malposed teeth; (4) avoid the problems which abnormal tooth and jaw relations create in later years when teeth are lost and the edentulous spaces need restoration.

Timing in Treatment

In planning the correction of a malocclusion one of the biggest problems has to do with timing of treatment.[9] At the outset I would make the point that a child with a malocclusion should be referred for orthodontic consultation at the time of recognition. This is advisable, not for the purpose of immediate treatment, but rather for the value of the continued observation which allows the orthodontist to gauge the growth of the patient, the development of the jaws and the eruption pattern of the teeth. In many malocclusions the pattern has been established at least at birth, and spontaneous correction is uncommon.

On the other hand, with occasional exceptions, as in some cases of mandibular prognathism, malrelations of the jaws do not tend to become progressively severe.

Malocclusions are seldom treated during the period of the deciduous dentition. Most malocclusions are treated after all the deciduous teeth have been lost. However, in recent years there has developed a renewed interest in early treatment, that is, during the period of the transitional or mixed dentition when both deciduous and permanent teeth are present (seven to 12 years). The advantage of this early treatment lies in the possibility of creating space in a crowded dentition during a period of fairly rapid or continued growth. An especially desirable period for initiating treatment is the parapuberal period, when growth is somewhat accelerated. Then the advantage stems not merely from the increase in the size of the jaws, but also from an apparently greater facility for adaptation in the tissues.

Successful Treatment and the Growth Potential

The successful outcome of treatment is closely linked with adequate growth and ultimately depends upon good health and nutrition. The orthodontist whose intuition or good judgment allows him to gauge the growth potentials of his patients is in a better position to manage his cases successfully. In the future it may be possible to base this judgment on more objective criteria. We know that skeletal growth is accelerated in the prepuberal period, but much remains to be done in working out the relation between the chronology of general skeletal growth and jaw growth. The work of Nanda[23] is an excellent beginning. It appears that skeletal age as determined from hand films[16] is reliable for the prediction of ultimate statural attainment.[2] A great advantage will have been gained for treatment planning in orthodontics if this kind of information can be used to predict the growth potential of the jaws.

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808 South Wood Street
Chicago 12, Illinois

THE FRACTURED INCISOR

BERNARD Z. RABINOWITCH, D.D.S.

It is hardly possible to estimate the frequency of injury to the anterior teeth. Accidents to the primary teeth occur most often as the infant attempts upright locomotion and, finding the foundation unsteady, bends forward and falls on his face. The greatest number of injuries occur to children with a maxillary protrusion. The damage is in proportion to the force and the angle of trauma.

In normal development the tendency is toward more alveolar growth in the anterior area in order to provide for the permanent incisors; therefore there should be no loss of space. Fractures of the primary incisors need not be a cause for too great concern. There may be exceptions in the instance of pre-existing habit syndromes or when the hereditary pattern shows large teeth in small jaws and/or a predisposition to malocclusion. Additional points to be considered may be (*a*) the effect of the injury or loss of tooth structure on speech; (*b*) the formation of a tongue or swallowing habit; and (*c*) the psychological problems of the child and parents.

INJURIES TO PRIMARY TEETH

Injuries to the primary teeth may be classified as follows:

1. Fracture of the enamel or slightly into dentin
2. Fractures into the dentin
3. Fractures into the pulp
4. Fractures of the root
5. Comminuted fractures
6. Displaced teeth

Fractures of the teeth are often associated with lacerations of the mouth. It is important to control hemorrhage as quickly as possible and close the wound as quickly as feasible. Consideration should be given to the need for tetanus prophylaxis and antibiotic therapy (Fig. 45).

Fractures of the enamel or slightly into the dentin are the most frequent type found in the primary dentition. Immediate attention should be given these, for they are not as harmless as is often thought. The seriousness depends upon the angle and force of the trauma. The pulp may have been severed at the apex, causing devitalization. The edema at the apex may strangulate the pulp and also cause devitalization. The hemorrhage may have penetrated the dentin, causing the tooth to

Fig. 45. Left central and lateral incisors totally displaced and right central incisor retruded as result of an acident in a child 4 years and 8 months old.

Fig. 46. Vital "black tooth" 6 weeks after a fall.

darken, which has often been taken to mean that the tooth is nonvital, but such is often not the case. It may mean merely that the hemoglobin is oxidized and decomposed with its typical "black and blue" reaction. If the blood supply is adequate and can reach the area, the color will disappear and the tooth may lighten. Sometimes the discoloration may remain and the tooth still retain its vitality (Fig. 46).

Roentgenograms should always be taken to determine the extent of the damage and saved for further comparative study. There should be a careful clinical examination; sharp edges may be smoothed and the parents and the patient calmed and reassured.

A fracture into the dentin, after the routine x-ray and clinical examination, usually requires sedative treatment and insulation from temperature changes with a suitable temporary guard or splint.

A fracture into the pulp must receive immediate attention. After the x-ray and clinical examinations have been made to determine the extent of injury one must determine whether or not the tooth can be saved. If the tooth can be saved, it must then be determined what type of pulp treatment should be instituted: viz., pulp capping, pulpotomy or root canal therapy (these will be described later). A tooth with an infected pulp must not be allowed to remain untreated, for, in addition to the general hazard of sepsis, a hypoplastic mark may be left on the permanent tooth bud.

Frequently accidents can also cause teeth to be forced back into the alveolar process. These are sometimes retruded to the extent that they

Fig. 47. Left central incisor re-erupting 4 weeks after injury.

are considered totally avulsed. However, the routine x-ray examination will disclose the position of the tooth. These teeth often grow out again, re-erupt, and may remain vital (Fig. 47). Loosened teeth should not be extracted too readily, since they often become firm again and remain vital. Those that are displaced far to the lingual or to the labial should be gently and slowly returned to their approximate original positions if they interfere with normal function. If possible, the tooth should be stabilized with a cast or wired fixation.

Primary teeth forced back into the alveolar process may cause dilaceration of the succeeding permanent bud. This is a condition of the tooth produced by displacement of the calcified portion from the tissues which formed it, development being continued after the normal position of the calcified part has been lost. Figure 48 shows a ground section of tooth removed at age 9½ because it could not erupt. Dilaceration of the tooth occurred at age 22 months, caused by an accident to the primary central displacing it violently into the alveolar process.

INJURIES TO PERMANENT TEETH

Surveys by numerous investigators have shown that the most common period when fractures of the permanent incisors occur is between the ages of eight and 10 years. It is at this age that the teeth emerge and erupt into position with the root either partially developed or just completing its development. It is at this period that the teeth often project out more than any other portion of the child's face. As the child falls with his mouth open to cry out, the incisor teeth are usually the first to strike the ground.

The management of the fractured tooth is important from the physical, esthetic and psychologic aspects. The prime consideration is the preservation of the tooth, and an explanation of this for parents and

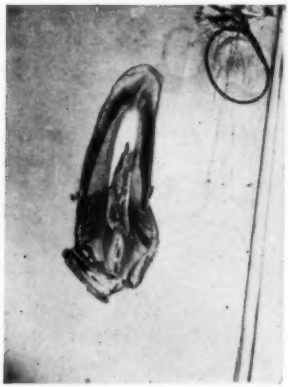

Fig. 48. Dilacerated tooth.

child is helpful in gaining cooperation. The injury must be seen as soon as possible. Delays in treatment may result in loss of vitality to the pulp. The history of the accident is helpful in determining the proper approach. Whereas previously, falls, drinking fountains, athletic games were most common causes, automobile accidents now are the leading causative factors.

Injuries to the permanent teeth may be classified as follows:

I. FRACTURES OF THE TOOTH

- A. Class I fracture—break in or through the enamel
- B. Class II fracture—break into the dentin not involving the pulp—the major group
- C. Class III fracture—break involving the pulp
- D. Class IV fracture—break involving the root
- E. Class V fracture—comminuted fracture

II. DISPLACEMENT OF TOOTH IN THE JAW

A. Partial avulsion
B. Total avulsion—with or without root fracture

Fractured permanent incisors can be classified in several ways, but the basis of all classifications is the extent and direction of the fracture (Fig. 49). The age of the patient, both chronological and physiological, must be considered. All traumatized teeth, whether fractured or not, should be x-rayed as soon as possible after the accident. Several films, at least three, taken at slightly different angles about 5 degrees apart, present a more comprehensive picture than a single exposure. In this manner one can make a three-dimensional study. After diagnosis these

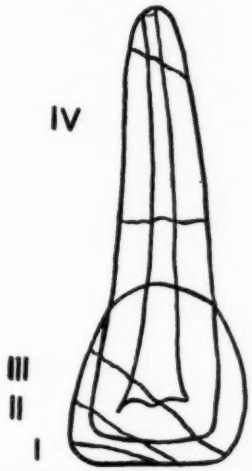

Fig. 49. Classification of tooth fractures.

films should be saved for future comparative study. Treatment is planned according to the degree of injury and directed toward the goal of having the tooth retained with complete vitality if possible. When vitality is lost, treatment is planned to maintain the tooth in position in a healthy state, thereby contributing to normal development of the face. All lacerations should be cared for at once and hemorrhage controlled.

Fractures of the Tooth

Class I Fracture. Usually a small part of the incisal is broken with only the enamel or at most a small amount of dentin involved.

Examine the roentgenograms to determine the extent of injury.

Test for vitality. Remember that responses may not be accurate for

several days after the shock of the initial trauma. Prolonged pain with an ice cone signifies a pulp hyperemia.

If an edge is very jagged, it may be smoothed slightly to prevent irritation. Normal wear will in a short time usually even the broken area until it will be hardly noticeable.

Class II Fracture. These are fractures into the dentin not involving the pulp. This is the main classification, over 50 per cent.

Examine the roentgenograms to determine the extent of injury.

Test for vitality.

Check for mobility and sensitivity to percussion and thermal changes.

Cover the fractured area with a sedative dressing and protect the tooth with a suitable guard or temporary splint.

Recent research has shown that zinc oxide and eugenol not only sterilizes dentin, but also is an obtundant for the relief of any pain present and because of its sealing qualities reduces irritation. If desired, a calcium hydroxide filling may be placed over the exposed dentin and then sealed with zinc oxide and eugenol. This sealing of the fractured area protects from irritation, temperature change and so on and may prevent pulpal death.

The temporary guard is used to cover the medicated dressing and also to retain the position of the tooth. When a mesial angle is broken off, the tooth, if unprotected, may drift, thereby creating a misalignment. Such misalignment is prevented by a temporary guard or splint. There are two main types:

For type 1, trim a plastic crown form or celluloid crown form to fit and fill with a suitable shade of plastic filling material. Place it over the fractured tooth that has been treated by a sedative dressing. Punch a hole in each incisal corner of the crown form so that the pressure of placement will be relieved.

In the previously contoured crown form place a mixture of zinc oxide and eugenol. This, however, discolors in time.

For type 2, contour a band of thin material, about 0.15 by 0.04 cm., around the fractured tooth. Pinch on the lingual and weld or solder. Make an incisal flap over the fractured area with a wider piece of band material, allowing the edges to remain open and permitting the flow of excess cement over the sedative dressing (Fig. 50).

After the tooth is no longer sensitive an esthetic semipermanent splint for long-range protection and maximum cosmetic effect may be placed. These will be described later.

Class III Fracture. These are fractures involving the pulp.

Examine the roentgenogram to determine the condition of the root. If no fracture or infection is present, a pulpotomy may be performed. If the initial injury has caused undue shock to the pulp tissue, palliative treatment allowing the pulp to recover is first undertaken. First cover the stump with a calcium salt such as dicalcium phosphate or calcium hydroxide sealed in the zinc oxide crown form. When the initial symptoms have subsided and the pulp recovers, a pulpotomy may be performed.

Treat the pulp as indicated (Fig. 51).

1. PARTIAL PULPOTOMY—usually done when the exposure is small, exposing merely a corner of the pulp.
   a. Anesthetize the tooth.
   b. Isolate the tooth and sterilize the area; where possible, place the rubber dam.
   c. With a sterile round bur penetrate the pulp, removing the superficial infected layer creating hemorrhage.
   d. After the blood has clotted remove the excess so that it will not decompose and discolor the tooth. Cover the clot with dicalcium phosphate or calcium hydroxide.
   e. Seal with zinc oxide and eugenol, avoiding pressure.
   f. Take postoperative x-ray films and dismiss the patient.

Fig. 50. Banded splint protecting fractured tooth.

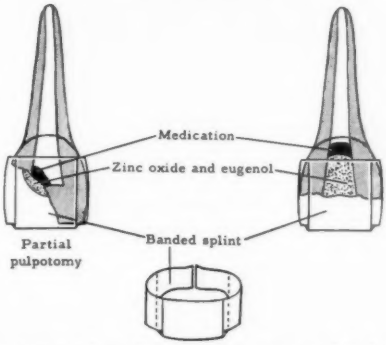

Fig. 51. Pulpotomy and partial pulpotomy protected by banded splint.

2. COMPLETE PULPOTOMY
   a. Anesthetize the tooth.
   b. Isolate the tooth and sterilize the area; where possible, place the rubber dam.
   c. With a large round bur open into the pulp chamber.
   d. With a clean sharp number 8 round bur revolving rapidly sever the bulbous portion of the pulp. Clean the remnants of the pulp with either bur or spoons.
   e. Control hemorrhage by absorbing with sterile cotton pellets and apply calcium hydroxide or dicalcium phosphate.
   f. Cover treatment with zinc oxide and eugenol, avoiding pressure.
   g. Take postoperative x-ray films and dismiss the patient.

h. After four to six weeks recall the patient and take roentgenograms. If the x-ray films and clinical study show normal healing, one may proceed with restoration of the tooth.

3. PULPECTOMY OR ROOT CANAL

If the tooth has become infected or if the trauma has been such as to sever the apical vessels, a pulpectomy must be attempted in order to preserve the integrity of the developing arch and face. On rare occasions one may see a tooth that has a pink coloration. This is symptomatic of an internal granuloma. The tooth with an internal granuloma may also on occasion be saved with root canal therapy.

a. Carefully study the roentgenograms for pulp anatomy and possible anomalies and extent of periapical involvement.
b. Some of these teeth have vital tissue remaining. When in doubt, anesthesia is indicated.
c. Isolate the tooth with the rubber dam.
d. Using a large round bur or diamond stone, enter into the pulp chamber.
e. Widen the opening for access and thoroughly cleanse the coronal portion. This prevents discoloration.
f. Ream and file the root canal and render it sterile by chemotherapy, antibiotics or electrosterilization.
g. Obtain two negative cultures.
h. Follow the trial point x-ray films and fill the root canals.
i. If deemed advisable, a root resection or curettage may be indicated.
j. Take a postoperative roentgenogram and dismiss the patient for about four to six weeks.

Fractures of the Root. Fractures of the root of a tooth need not be too alarming, but must receive immediate attention. Routine roentgenograms should be taken for diagnosis and future observation. The tooth that has a fractured root should be immobilized at once to prevent movement and yet not interfere with occlusion, thus allowing for healing of the fracture.

There are several methods of splinting whereby immobilization may be obtained: (*a*) wiring of injured teeth to adjacent teeth, (*b*) cementing and/or wiring an acrylic splint to injured and adjacent teeth, (*c*) placing a cast of surgical cement around the injured tooth and adjacent teeth.

If there has been no separation of the tooth fragments, the healing may take place by recalcification when the pulp vitality is retained. If separation of the fragments occurs, there may be a fibrous union, osteocementum union, or combination of both (Fig. 52). When the apical fragment is small or merely the tip of the root, it is commonly found to be resorbed. When the fracture is in the cervical third, mobility cannot be controlled, and often these teeth may not be retained and are resorbed.

In the event that clinical and x-ray study shows a periapical radiolucency, apicoectomy may be indicated. At this time the fractured root tip is removed. Apicoectomy is based on the premise that the root end of the infected teeth is an irritant and the periapical bone is unable to maintain its integrity in the presence of an irritant. The elimination of all sources of irritation is the aim of the operation.

Comminuted Fractured Incisors. Many times children present for treatment with anterior teeth fractured in several places. Some of these are compound, some are comminuted, and some are combinations of both. We used regrettably to order their removal. This resulted in psychic damage as well as physiologic and developmental interference. After observing the resultant damage following the early removal of a permanent tooth from children an attempt was made to preserve selected cases in which it was thought healing could be accomplished and some of the larger fragments cemented to the root portion and to each other by the formation of secondary dentin. This technique has been successfully used in selected cases:

Fig. 52. Root fracture caused by football accident. Coronal and apical pieces united by osteocementum. Note the pulp filled by "nature's root canal."

a. X-ray the tooth to determine extent of the damage. Three angles are taken 5 degrees apart to give a three-dimensional effect.
b. Give a local anesthetic.
c. Wash the tooth and surrounding area with normal saline solution, removing the debris.
d. Make a pinch band, solder or weld, and cement around the tooth crown, thereby binding all loose fragments together (Fig. 53).
e. The rubber dam can rarely be used. The area is cleansed by liberal application of tincture of merthiolate or a similar agent.
f. Perform pulpotomy. Carefully remove fragments that cannot be successfully retained. Place a temporary restoration.
g. X-ray films are taken for study and for future reference.
h. The patient is advised to return in four to six weeks for observation and x-ray examination.
i. This operation may be considered successful when the band is removed and the original fragments are firm under acceptable pressures and when the x-ray film shows evidence of secondary dentin obliterating the lines of the original fracture (Fig. 54).
j. Semipermanent restoration may be placed. One must remember that the laws of leverage will not allow for excessive pressures without refracturing the tooth (Figs. 55, 56).

Several principles should be adhered to for successful treatment in this type of case: (1) The tooth must retain its vitality. (2) The fragments to be retained must extend to and below the area of secondary dentin (calyx) formation. (3) The tooth must be restorable.

Fig. 53. Fig. 54.

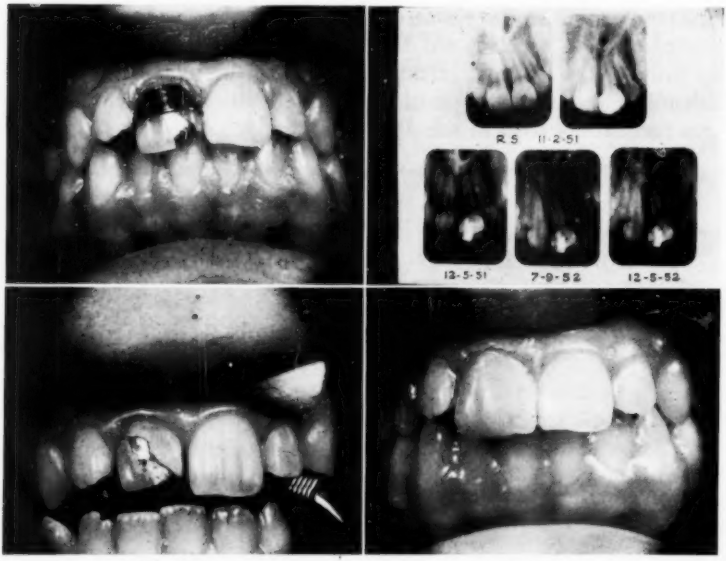

Fig. 55. Fig. 56.

Fig. 53. Fragments of a tooth with comminuted fractures held together by pinch band. Pulpotomy performed about 7 months prior to picture.

Fig. 54. Comminuted fracture of central incisor—5 pieces. 11/2/1951, Preoperative and postoperative roentgenograms. 12/5/1951, Check-up roentgenogram with evidence of secondary dentin calyx. 7/9/1952, Check-up roentgenogram showing large secondary dentin bridge. 12/5/1952, Check-up roentgenogram.

Fig. 55. Preparation of tooth shown in Figure 54. To receive a more esthetic restoration. Note 2 parts of the crown which are held together by secondary dentin.

Fig. 56. Gold-backed plastic crown of case shown in Figures 53, 54 and 55. This is a semipermanent esthetic restoration.

In selected cases in which these criteria were followed, teeth that it had been previously considered advisable to extract were maintained.

Displacement of Teeth in the Jaw

As was mentioned above, the damage is in proportion to the force and angle of trauma. This may sometimes account for displacement of the tooth with or without fracture of the tooth.

Partial Avulsion. In cases of partial avulsion there is often the pos-

sibility that vitality may be maintained. When this is so, the tooth is usually displaced lingually or labially with the apex acting as the fulcrum of the movement. If this does not interfere with occlusion, the tooth should be allowed to rest so that the symptoms of pain and shock may be allowed to abate. After a period of about a week the tooth may be repositioned with slow steady pressures and then stabilized by one of several techniques that will be described. If the tooth interferes with occlusion, it may be repositioned to its intended place in the arch under gentle steady pressures. These teeth may lose their vitality. When this occurs, it is assumed that the apical pulpal fibers have been severed. Root canal treatment may allow the tooth to be retained.

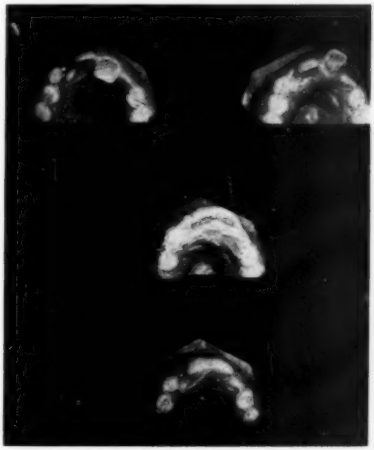

Fig. 57. Cast to immobilize avulsed tooth.

a. X-ray for the possibility of fractured root.
b. With extremely gentle pressures return the tooth to as close to its original position as possible. This is a slow, time-consuming operation.
c. Place a cast or splint to immobilize and maintain position.
d. X-ray for study and future reference.
e. The patient is sedated and advised to return in seven to 10 days. A semisolid diet rich in vitamins and minerals is prescribed.
f. When the patient returns, the splint or cast is removed, and the tooth is x-rayed and tested for vitality. One must remember that the tooth has suffered extreme shock and may not respond to present methods of vitality testing for some time (Fig. 57).

In some cases the traumatic irritation has stimulated secondary dentin formation to the point at which calcification will have completely obliterated the pulp canal.

When partial avulsion occurs in which the tooth has been displaced somewhat out of the socket, the above precautions should be observed.

If it can be retruded somewhat, yet without extreme pressures, and immobilized, vitality may be retained. If the original position is not completely obtained, slight grinding to reduce the incisal, after the tooth has been rested and recovered, may be done. These teeth must be observed so that, if they are devitalized, root canal therapy may be instituted before the area becomes infected.

Cases of partial avulsion in which the tooth is retruded back into the alveolus occur in great numbers in the primary dentition, but only rarely in the permanent teeth. The only thing one should do is to cleanse the debris, close the lacerations, and stop the hemorrhage. Then x-ray for study. The patient is recalled at regular intervals to observe re-eruption and for x-ray pictures.

Teeth that are only slightly displaced are almost too numerous to count, yet not severe enough to require immobilization. These need be given no special attention other than routine x-ray and observation and patient advice.

Total Avulsion or Displacement. There are an increasing number of accidents wherein the anterior permanent teeth are completely displaced. If these teeth can be replanted successfully, they help maintain the integrity of the developing arch and maintain the facial growth until the normal adult features are attained. There is also an esthetic and psychological factor which should not be minimized. Several principles should be adhered to for successful replantation.

a. The avulsed tooth and socket should be treated as any fractured bone, reduced and immobilized.
b. The tooth should have its root canal sealed to prevent infection.
c. The tooth should be cleansed of debris, but the periodontal fibers should not be removed. This allows for possible readaptation of these fibers.
d. The tooth must be replanted and immobilized in a fresh healthy socket.
e. No excess pressures are to be exerted either in the replantation procedure or by traumatic occlusion.

An important phase of the treatment, perhaps the most important, is adequate explanation to parents and patient as an aid in cooperation both in treatment and home care.

TECHNIQUE OF REPLANTATION.

a. Cleanse the tooth in warm saline solution. Do not scrape off the periodontal fibers.
b. X-ray the socket to determine the possibility of a fractured root end, which should be removed.
c. Remove debris and loose bony fragments. Do not disturb the periosteum. When the labial plate is fractured, try to preserve both bone and periosteum. Control hemorrhage and suture lacerations.
d. Do the root canal filling on the avulsed tooth.
e. Freshen the blood clot and then gently but firmly replace the tooth into the socket as far as it will go. Do not force beyond limits of inflamed tissue, or necrosis and resorption may result. Antibiotics may be instituted.

f. Splint into position and immobilize with a cast of periodontal (surgical) cement into which are incorporated strips of gauze ¼ inch wide for binding and strength. These are wrapped around all available teeth at the gingival and compressed into the interproximal. The patient is instructed to close the teeth to check for occlusal interference. Hold the replanted tooth firmly in position until the cast is set.
g. Roentgenograms are taken for study and future reference.
h. Tetanus inoculation is advised when indicated, as are the antibiotics.
i. The patient is sedated and advised to return in seven to 10 days. A semisolid diet high in ascorbic acid is prescribed.
j. When the patient returns, the cast may be removed. Roentgenograms are taken and occlusion is checked to avoid trauma. If desirable for any reason, a cast may be reapplied. A more esthetic splint may be applied by molding quick-setting acrylic over a cast made from a wax impression. This is wired and/or cemented to the adjacent teeth.

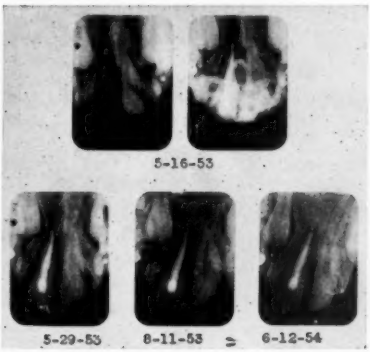

Fig. 58. Totally displaced central incisor. 5/16/1953, Preoperative and postoperative roentgenograms. 5/29/1953, Check-up roentgenogram after removal of cast. 8/11/1953 and 6/12/1954, Check-up roentgenograms.

As the inflammation subsides the tooth will resume close to its normal position in the socket either by ankylosis or combination of ankylosis and periodontal reattachment. If the tooth is left long, it may later be ground down to match the adjacent tooth.

A cast is preferred over wiring, since it seems to immobilize the replanted tooth better and to reduce the incidence of traumatic occlusion (Fig. 58).

Because of the possible deleterious effect on the developing permanent buds no attempts have been reported on replantation of primary teeth.

Unfortunately not all teeth can be saved, and some teeth must be lost. In the permanent dentition this space must be preserved by a suitable appliance to preserve the integrity of the arches and face.

SPLINTS, CROWNS AND ESTHETIC RESTORATIONS

Temporary Splints

These are used primarily for protection.

Crown Forms. These are celluloid, plastic, stainless steel or gold.

CELLULOID OR PLASTIC. Over a sedative dressing these may be contoured and cemented with a tooth shade plastic material or zinc oxide and eugenol. The esthetics is only temporary, since these readily discolor.

STAINLESS STEEL OR GOLD. These may have the labial cut out for better esthetics. They are cemented on over a sedative dressing.

Banded Splints. With thin band material, 0.15 by 0.04 cm., make a pinch band around the tooth, and solder or weld. Attach another piece of wider material from the labial to the lingual over the incisal fractured area. This is cemented on over a sedative dressing.

These restorations may be used as emergency protective measures and kept under observation for at least three to six months (Fig. 50).

Semipermanent Restorations

These are placed after the emergency protective covering has been in place for at least three to six months and the tooth remains vital and comfortable with no history of pain. X-ray films and clinical examination allow one to decide when a more esthetic restoration is deemed advisable.

Plastic Corners. The fractured area is prepared with adequate retention on incisal, labial, lingual and approximal with good retentive undercuts. If deemed advisable, pins may be used for added retention (Figs. 59, 60).

Plastic Crown. This gives esthetic restoration with the advantage of good shading and color match. However, it is slightly oversize, and there is color change. These may have a gold backing for added strength and resistance to abrasion.

Three-Quarter Crown. This is probably the most satisfactory restoration when conditions are favorable and the fracture not too large. Acrylic, silicate or porcelain may be used to supply the missing tooth structure (Fig. 61).

Permanent Restorations

Three-Quarter Crown. The only disadvantage is some lack of esthetics because a little gold will show.

Jacket Crown. These should not be made until the tooth has ma-

tured and the pulp filled in and receded and the face has achieved its adult size and form. This rarely occurs before the age of 18 years.

PLASTIC JACKET CROWN. Though at first the color match is excellent, the color changes because of absorption of mouth fluids. There is usually some gingival irritation.

Fig. 59.

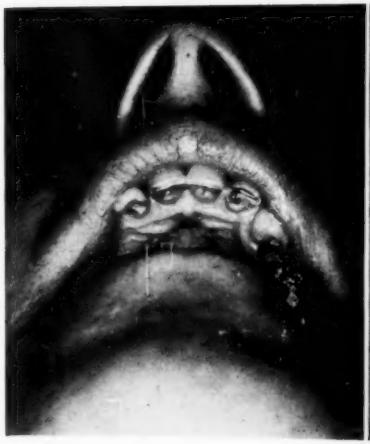

Fig. 60.

Fig. 59. Mesial angle fracture of left lateral incisor prepared to receive plastic corner restoration. Note pin retention.

Fig. 60. Completed restoration of plastic corner.

Fig. 61. Two three-quarter crown restorations.

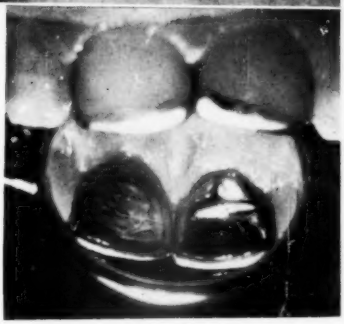

Fig. 61.

PORCELAIN JACKET CROWN. The shade is excellent. Tissue tolerance is good. However, these are easily breakable.

PERMADENT CROWN. This is a new type of restoration utilizing cast platinum as a core and a special type of porcelain that fuses to the platinum. The esthetics is good and they are less susceptible to breakage and could be used in some selected cases at an earlier age because the preparation of the tooth may be shallower.

PREVENTION OF INJURIES

We have spent all this time discussing care of fractured incisors and how to save them, but there are preventive measures which may be instituted. Protruding teeth should be corrected at an early age before a fall or blow causes a fracture. This may have to be done in some cases even if it means that there will have to be a second stage of orthodontic treatment. Mouthpieces may be molded to protect teeth during contact sports. Since these have been used more the incidence of traumatic injury to teeth due to contact sports has materially decreased.

CONCLUSION

Since it is inevitable that many accidents will occur in which teeth will be fractured, the dentist must be aware of a complete procedure so that he may proficiently care for these problems in collaboration with the physician. It is within the scope of every practice, and a successful treatment is dependent upon a thorough study of the problems involved and an awareness of the required treatment. It is important that the attention be given promptly; only then may many unfortunate sequelae be prevented.

5720 Wilshire Boulevard
Los Angeles 36, California

CLEFT LIP AND CLEFT PALATE

Perspectives in Management

EDWARD F. LIS, M.D.

SAMUEL PRUZANSKY, D.D.S.

HERBERT KOEPP-BAKER, PH.D.

HERBERT R. KOBES, M.D.

Among congenital defects, cleft lip and palate is unique. It has been described as a deformity that can be "seen, felt, and heard."[23] Not many defects are so disturbing to parents and so handicapping to the child, and yet few are as amenable to correction by properly planned treatment. Habilitation, however, is not simple, but often complex. It is not of short duration, but extends frequently from birth through adolescence. It is no longer the surgeon's task alone, but involves the integrated efforts of the best in medicine, dentistry and special education. Although many specialists may be involved in the long-range treatment program, the child's physician remains a central figure and is significant to the over-all planned effort.

This clinic is designed to serve two purposes: First, to provide the family physician or pediatrician with a broad orientation in problems facing the family, the child and the community. Such orientation is required for a better understanding of the need and the sequence of the various therapeutic events that may be required. Secondly, to provide the child's physician with specific information essential to his role as family counselor and member of the habilitation team. The

From the Cleft Palate Center and Training Program, University of Illinois Professional Colleges, Chicago, Illinois.

frequency of this anomaly is sufficiently high to warrant such elaboration.

GENERAL ORIENTATION

Frequency of Cleft Defects

In most medical practices relatively few children are found with cleft lip or cleft palate. Cumulative statistics, however, indicate a considerable and increasing number in the general population. In the United States, utilizing a variety of records, it has been estimated that one child is born with this defect in every 550 to 1000 live births.[23] In local studies, using birth certificates as a guide, Wisconsin has a reported incidence of 1:770[25] and Pennsylvania 1:800.[11] The six New England states anticipate approximately 270 babies with clefts each year;[4] Illinois approximately 250.[13] In 1952, throughout the country, approximately 5000 were born with cleft lip, cleft palate, or both.[23]

These statistical data probably represent a conservative estimate of the true incidence. Small clefts of the soft palate often go undetected until a feeding problem is presented. Submucous clefts of the palate are more difficult to diagnose early and are detected usually in the more active stages of language development or after an adenoidectomy, which precipitates speech unintelligibility. The increasing number and proportion of cleft lip and palate in the population may be attributed to the increased birth rate; to improved methods of reporting; to the greater survival rate of infants and possibly by the proliferation of such individuals.

For reasons not at all understood there are some enigmas in the occurrence of cleft defects. Males have more clefts of the lip, with or without cleft palate; females, more cleft palate in isolation to lip defects. Clefts of the left side of the lip are much more frequent, followed by bilateral clefts; less common are right-sided clefts.[7] In the colored population all clefts are about one half the incidence of the white population.[5] In certain areas in the United States and elsewhere where consanguinity is common, clefts occur in greater incidence.

Changing Trends in Management

By tradition the management of cleft lip and palate was entirely entrusted to the surgeon, and the responsibility of planning the entire program of care, extending over a number of years, was delegated to one man. The surgical goal was obvious—to repair the lip and palate as soon as possible in order to obtain an optimal cosmetic and func-

tional result. That such end results were often obtained was in large measure a credit to the surgeon's skill, perseverance and ingenuity.

Surgery, however, did not always succeed in achieving all its objectives. Results which seemed successful shortly after operation sometimes developed into failures. Throughout the country over a period of years a number of such surgical failures accumulated. These were characterized by disfigured faces, underdeveloped maxillas, distorted dentition and, worst of all, the inability to speak understandably (Fig. 62).

Fig. 62. Characteristic facial, palatal and dental deformities resulting from traumatic and excessive surgical procedures for the repair of cleft lip and palate.

When normal speech failed to develop, the parents were counseled to find a good speech teacher who would provide the necessary "lessons" and blowing exercises to develop the muscles of the soft palate. If the teeth were malposed and the upper jaw poorly developed, the orthodontist was supposed to correct the defect. If all such attempts failed, the prosthodontist was assigned the task of fitting the child with a prosthetic speech appliance that would replace the missing teeth, plump the upper lip, and by its extension into the nasopharynx substitute for the short, nonfunctional soft palate. Then there was always the new surgical procedure that could be tried as a last resort.

Therapy was piecemeal and based more on empiricism than on science. In their quest for proper treatment parents and child were

shunted from one specialist to the next. They encountered an assembly line of consultants, each isolated from the others by barriers of specialistic interest and individual brands of jargon. These efforts, in terms of therapy for the patient, were often disjointed and sometimes at cross-purpose. All this led to hit-or-miss care for the child with inconsistent results, and to financial and psychological strain for the family. Significantly, in the midst of all this the child was developing with little attention paid to his social and emotional maturation.

A number of separate events served to change this picture, at least in some areas. First, it must be recognized that the frustrations encountered in treating these children were shared by the professions. Physicians, dentists, speech pathologists, public health workers and others concerned with the problem recognized the futility of their individual contributions when unrelated to the total care of the child. Out of this experience representatives of several professions met in joint deliberation to unite their separate efforts in an integrated program. A forum for the exchange of these ideas was found in the establishment of The American Association for Cleft Palate Rehabilitation.

In the past decade the information derived from research on the growth of the facial bones in man was applied to the study of the cleft palate. As a result it was determined that poorly conceived, ill-timed and excessive surgical procedures were often the cause of failure of the facial bones to develop properly in patients with cleft palate.[10, 24] In contrast, a group of cleft palate subjects who were never operated upon demonstrated patterns of facial growth resembling the normal.[10] Consequently there developed a movement to delay surgical repair of the palate until four to six years of age in order to minimize the deleterious effects upon facial growth. Presumably, proponents of this school of thought assumed that all operations upon the palate posed a definite impediment to the natural growth processes of the face. By delaying operation they hoped to minimize the retardation of facial growth. In part, this viewpoint was justified, but as a generalization it did not take into account certain facts. For instance, many cases were recorded in which facial growth and speech developed within normal limits despite the fact that the palate was repaired before the child was one year of age.

It was clear that too many variables were operating to permit the formulation of simple and all-inclusive rules such as the fixing of the age at which all clefts of the palate should be repaired. Factors beyond the control of the surgeon's skill, such as variations in the severity of the original deformity and differences between children as children, could influence the success or failure of the surgical effort. The task was to identify and analyze these differences among patients in order to prescribe treatment on an individual basis.

A Center for Training and Research

In 1948 The Children's Bureau, now a part of the Department of Health, Education and Welfare, recognized that there was a national need for training interested specialists in all dimensions of the cleft palate person's need. To promote such knowledge, foster research and train professional personnel, The Children's Bureau provided a grant for the establishment of a Cleft Palate Center and Training Program at the University of Illinois.

In order to provide fundamental information supportive to the objectives of this Center, a longitudinal growth study of newborn infants with cleft lip and palate was begun in 1949. The research program was designed primarily to solve a number of clinical problems. Simply stated, these problems resolved into four questions: (1) What should be done for a child with a cleft lip and palate? (2) How should it be done? (3) When? (4) Does the treatment vary from child to child?

It became clear that the answers to these questions would depend in large part upon our knowledge of (*a*) how the child with a cleft differs from a normal child, and (*b*) the individual potentialities for growth and development of these children.

The longitudinal study now includes data on 500 different children. These patients represent the surgical experience of 15 different plastic surgeons. Not all these surgeons share similar views as to the indications for surgery, the timing of surgery or the kind of surgical technique to be used. Altogether, this investigation represents an efficient and convenient means for collecting and analyzing data that have heretofore been dispersed. It is these research data and the clinical experiences derived in these studies that provide a basis for much of this clinic.

ANATOMY OF CLEFT LIP AND PALATE

Not all clefts are alike. Cleft lip and cleft palate does not present a single fixed clinical entity which is subject to generalizations of description and classification. Not only are there different kinds of clefts, but even within a single kind there exist many variations. Because of such variation, it follows that no rigid formula of treatment applicable to all clefts should be expected.

Cleft Lip

Cleft lip is more than a defect in the lip alone. It is an anomaly complex with demonstrable defects in the nasal alar cartilages, the nasal cavity and the alveolar ridge on the affected side. A cleft of the lip may be complete, extending from the vermilion border to the floor

of the nose, or it may be incomplete. Various degress of incompleteness are recognized. Minimal defects occur in which the cleft extends only to the vermilion border. In others the defect may extend up to the nose as a submucous cleft in the muscle band, bridged only by mucous membrane, skin and fibrous connective tissue. The nasal alar cartilage on the side of the cleft is displaced and flattened to a greater or less

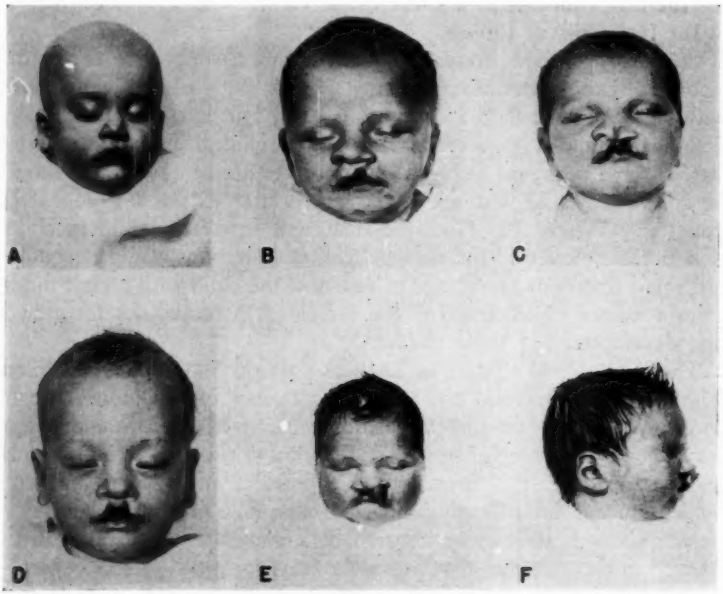

Fig. 63. Varieties of unoperated clefts of the lip. *A*, Incomplete cleft of the lip on the left side extending beyond the vermilion border. Note relative symmetry of the nasal alae. *B*, More complete unilateral cleft of the lip. *C*, Complete unilateral cleft of the lip extending into the floor of the nose. Note the extreme flattening of the alar cartilage on the side of the cleft and the deviation of the tip of the nose toward the normal side. *D*, Symmetrical incomplete bilateral cleft of the lip. *E*, Complete bilateral cleft of the lip. *F*, Profile view of the patient in *E*. The forward projection of the prolabium and premaxilla is characteristic of this deformity.

degree, depending on the extent and width of the cleft. The tip of the nose is deviated toward the noncleft side (Fig. 63).

The cleft in the lip may occur bilaterally and may be symmetrical or asymmetrical, depending on whether the lip is equally cleft on both sides. In the complete bilateral cleft lip a median portion of the lip is isolated in the midline and remains attached to the premaxilla and to the columella. This portion of the lip contains the philtrum. The premaxilla protrudes considerably forward of the facial profile and is attached to the nasal septum (Fig. 63, *E*, *F*).

There is a direct relation between the extent of the cleft in the lip and that in the alveolar process. The more complete the cleft in the lip, the greater the defect in the alveolar process and the greater the incidence of missing or malposed dental units in the line of the cleft. Also encountered in the line of the cleft are supernumerary or malformed teeth.

Cleft Lip and Cleft Palate

Clefts of the lip and palate may be unilateral or bilateral. They may be complete or incomplete. In a complete unilateral cleft of the lip

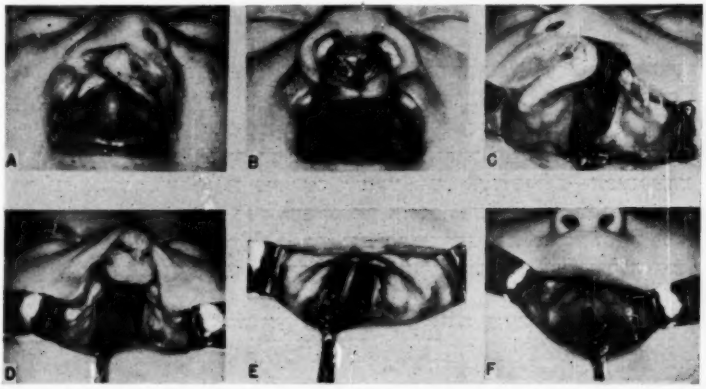

Fig. 64. Varieties of clefts of the hard and soft palates. *A*, Unilateral cleft of the alveolar process with intact hard and soft palates. *B*, Incomplete bilateral cleft of the lip with intact hard and soft palates. *C*, Complete unilateral cleft of the lip, hard and soft palates. *D*, Complete bilateral cleft of the lip, hard and soft palates. *E*, Cleft of the hard and soft palates without involvement of the lip or alveolar process. *F*, Cleft limited to the soft palate.

and palate a direct communication exists between the oral and nasal cavity on the cleft side. The nasal septum is attached and deviated to the palatal process on the opposite side (Fig. 64).

It is important to observe that a wide range of variation exists in each category. Some unilateral clefts exhibit wide separation of the palatal processes; in others this is less so; and in some there is actual overlap of segments (Fig. 65).

The striking features of the bilateral cleft lip and palate are the direct communication between both nasal chambers and the oral cavity, and the stalklike nasal septum bearing a mobile premaxilla. The premaxilla may be small or large, symmetrical or asymmetrical. Such variation may affect the amount of tissue available for repair and is related to the number of incisor teeth erupting into this segment (Fig. 66).

Isolated Cleft Palate

In this category neither the lip nor the alveolar process is involved. The cleft may extend only through the soft palate or the soft and hard palates, but not the hard palate alone (Fig. 67). This observation is in accord with the findings that fusion of the hard and soft palates proceeds from in front backwards.

In the extreme form the cleft may extend anteriorly as far forward as the nasopalatine foramen. In such instances there is a direct com-

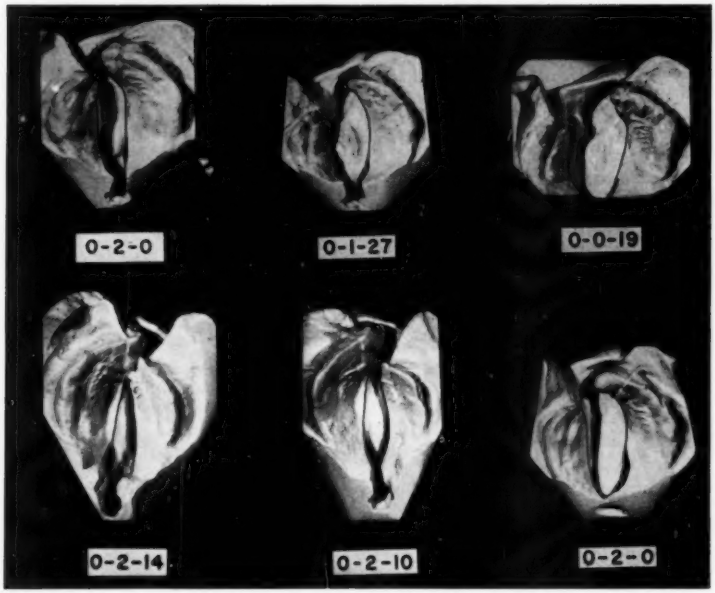

Fig. 65. Plaster cast reproductions of several varieties of unoperated complete unilateral clefts of the lip and palate.

munication between both nasal chambers and the oral cavity. The outline of the cleft may be wide or narrow and long or short.

Analysis of Variations

In surveying the range of variables encountered between different kinds of clefts and within a single kind, the following factors emerge as important in the analysis of the defect.[18]

Adequacy of Parts. The first thought that occurs when one sees a cleft in a newborn child is whether or not something is missing. Does the cleft represent a nonunion of parts that are intrinsically adequate,

or are the parts, per se, inadequate or even in excess? If the individual parts are hypoplastic, any attempt to bring these parts into direct apposition by surgical means will produce a total structure that is deficient.

Distortion of Parts. Muscle tissue develops and begins to contract in utero during the early calcification and growth of bone. The ab-

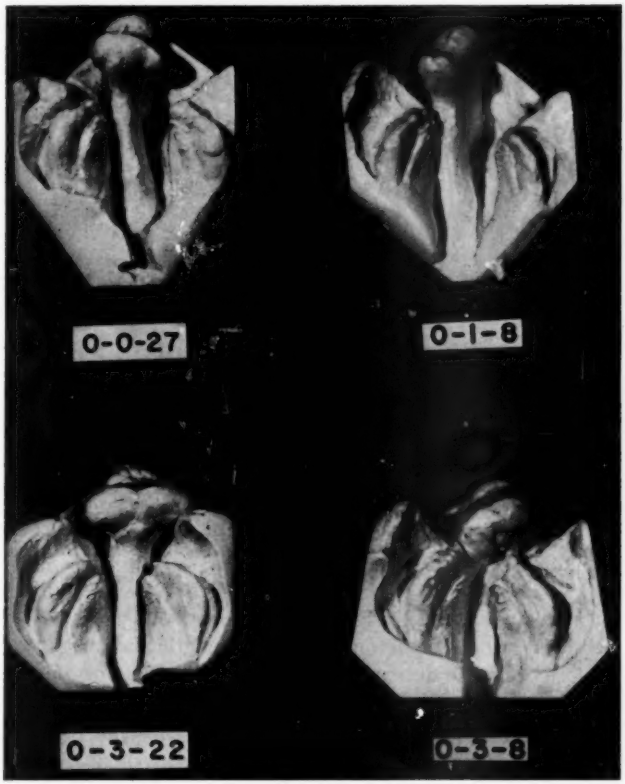

Fig. 66. Plaster cast reproductions of several varieties of unoperated complete bilateral clefts of the lip and palate. Note variation in size, shape and position of the premaxilla.

normal directions of muscle pull in a cleft lip or cleft soft palate may deform the bony and cartilaginous structures during their most prolific periods of growth. The degree of distortion of parts may affect the process of surgical reconstruction.

Relations of Parts in Space. The action of the tongue, cheeks, palatal and pharyngeal muscles on the separated bony palatal segments con-

tributes to the displacement of these segments. Similarly, the extent of such displacement may affect the ease of surgical reconstruction.

Relation to Adjacent Structures. The individual ability to compensate for a localized anomaly depends not only upon the severity of the defect itself, but also upon anatomic and functional variations in

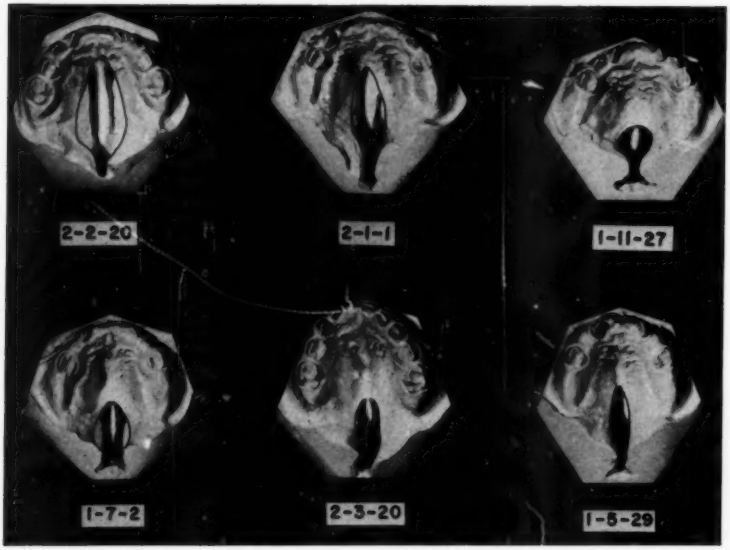

Fig. 67. Plaster cast reproductions of several varieties of unoperated cleft palate. The cleft may extend, in varying degrees, as far forward as the nasal palatine foramen. In its lateral dimensions the cleft may be wide or narrow.

adjoining structures. The capacity of a repaired soft palate to function effectively is dependent not only upon its length and range of elevation, but also upon the dimensions of the nasopharynx and the function of the accessory pharyngeal muscles. For these reasons the preoperative evaluation of adjacent structures is an essential aspect of the diagnosis.

PATHOPHYSIOLOGY OF CLEFT PALATE

Breathing is not usually difficult in infants with cleft lip and palate in the absence of respiratory tract anomalies, unless the nares are markedly flattened or the tongue mass rests in the nasal cavity. In isolated cleft palate the frequently associated mandibular micrognathia produces obstruction of the pharyngeal airway by the retroposed tongue, and severe respiratory distress manifest by chest retractions, hyperpnea, dyspnea, cyanosis and irritability may be present. The quality of the infant's cry is usually normal in the absence of obstructive symptoms.

Swallowing patterns are altered invariably because of the absence of an anatomic and functional separation from the nasal cavity and by the altered tongue dynamics imposed by the anomalous environment. Deglutition is accomplished without the assistance of the positive pressures available in an intact oral cavity and is dependent on positioning and gravity, especially during infant feeding.

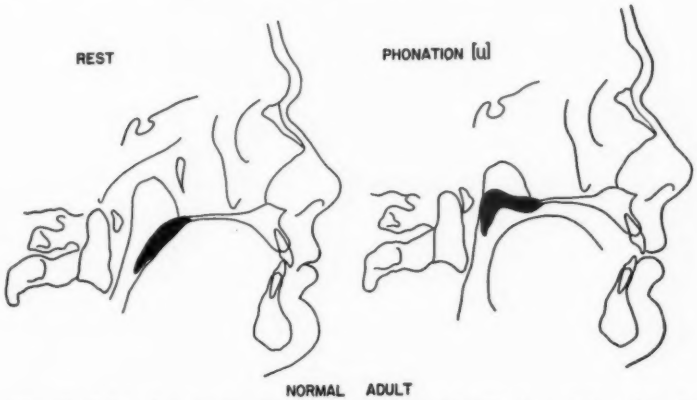

Fig. 68. Palatopharyngeal valving in a normal adult. Tracings of lateral cephalometric roentgenograms reveal the position of the soft palate within the nasopharynx. At rest the soft palate, outlined in black, rests on the dorsum of the tongue. During the sustained phonation of the vowel *u* the soft palate elevates to contact the posterior wall of the nasal pharynx.

Feeding is further complicated by many factors: the inability to seal off the nipple if the lip or alveolus is cleft, the inability to suck efficiently, and the disruption of normal deglutition.

Eustachian Tube Function

The higher incidence of middle ear infection in children with cleft palate suggests some disturbance in and around the eustachian orifice that is related to the cleft. Whether this is an anatomic variation or is due to impaired tubal function with possible increased contamination by the contents of the oral cavity remains to be demonstrated.

Speech

Speech is not a basic physiological activity, but is an imitative, learned accomplishment dependent on the vegetative functions of sucking, chewing, swallowing and breathing. Aberrations in the structure or function of the vegetative activities can be expected to alter the speech performance. Since the cleft of the palate prevents the usual

bony and muscular separation of the oral and nasal cavities, control of the air stream during speech becomes impossible. It is this disturbance which is basic to the speech defect of cleft palate.

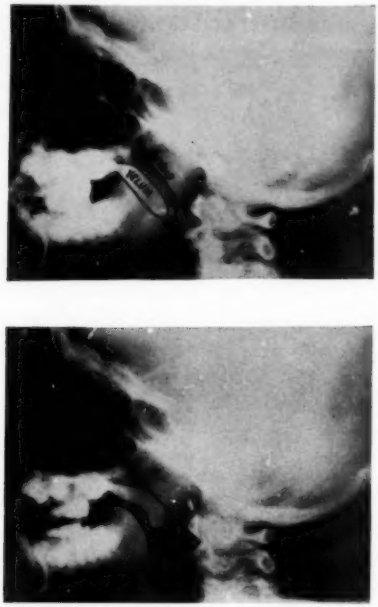

Fig. 69. Palatopharyngeal valving in a child with an intact adenoid mass. Note that during the sustained phonation of the vowel *u*, as revealed in the lower illustration, the soft palate makes contact against the adenoid projection into the nasopharynx rather than with the posterior wall of the pharynx.

Other factors which contribute to the speech patterns of cleft palate arise from the abnormal tongue postures and deformities of the lips, jaws, palatal vault and dentition.

Palatopharyngeal Action

In a normal person the muscles of the soft palate and pharynx are capable of integrated and repetitive movements which variably open and close the nasopharyngeal airway. The soft palate moves upward and backward toward the posterior wall of the nasopharynx (Fig. 68). At the same time there is medial movement of the lateral pharyngeal muscles and a less well defined activity of the posterior superior pharyngeal constrictor muscle. During speech these contractions are fairly rapid, while in deglutition they are slower and of longer duration.

In childhood the valving action of the soft palate is such that it

contacts the posterior pharyngeal wall in the region of the adenoid mass (Fig. 69). In later years, with further downward growth of the palate, the contact area is below the adenoid protuberance. Thus in childhood the adenoid has a passive role in palatopharyngeal closure. This role is not necessarily critical except for a child with a potential for palatopharyngeal incompetence.

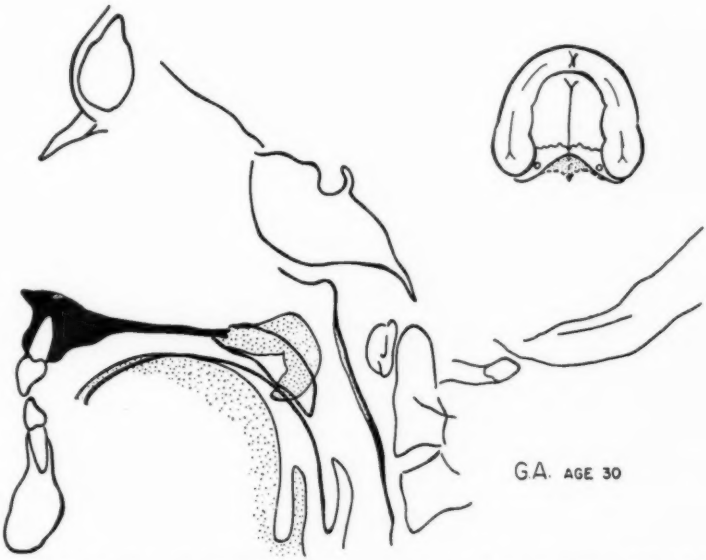

Fig. 70. Tracings of midsagittal laminagrams of an adult with congenital palatal insufficiency and submucous cleft palate. During the sustained phonation of the vowel *u* the soft palate is incapable of contacting the posterior pharyngeal wall and allows for the escape of air through the nose. The stippled area of the posterior border of the hard palate in the smaller drawing indicates the extent of the submucous defect in the hard palate.

In the unrepaired cleft palate normal valving function is not possible except in unusual anatomic circumstances. The inability to effect nasopharyngeal closure is not entirely due to the "hole" in the soft palate, but may also be associated with a congenitally foreshortened soft and hard palate (Fig. 70). It has also been demonstrated that the lateral dimensions of the nasopharynx in cleft palate are wider than normal, and this further complicates the mechanics of valving.[28] In some instances marked compensatory activity of the lateral and posterior pharyngeal muscles may be seen. On phonation or gagging, the lateral muscles may move toward the midline, while the posterior muscles may reveal a protruding ridge below the adenoid mass (Passavant's bar).

Sometimes such muscular activities may compensate successfully if inadequate palatal length persists after operation (Fig. 71).

Briefly, the requirements for effective palatopharyngeal function can be summarized as follows: (1) intact palate with adequate length, (2) mobile soft palate with upward and backward movement, (3) normal configuration of pharynx, (4) appropriate movements of lateral (and posterior) pharyngeal muscles.

Palatopharyngeal Incompetence

The condition in which the soft palate and associated pharyngeal muscles fail to separate the nasopharynx from the oropharynx has been

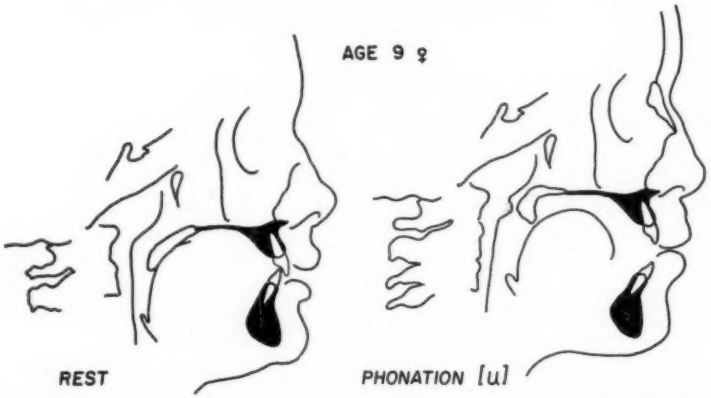

Fig. 71. Congenital palatal pharyngeal incompetence in a young child revealing compensatory activity of Passavant's bar during the sustained phonation of the vowel *u*. The formation of Passavant's bar on the posterior wall of the pharynx is indicated in the stippled area.

defined as palatal insufficiency by Dorrance[6] and more recently as palatopharyngeal incompetence by Calnan.[3] The latter term is preferable because it encompasses a larger variety of congenital and acquired clinical entities which share this disturbance in common with cleft palate.

Congenital disturbances include hypoplasia of the hard or soft palate, submucous cleft palate, anomalies of the nasopharyngeal spaces as in occipitalization of the atlas, and neuromuscular disorders secondary to congenital intracranial defects. The acquired factors include local derangements due to trauma, infection or malignancy, and neuromotor disorders such as bulbar poliomyelitis and brain injury.

Symptoms.

1. Hypernasal speech defect particularly evident in the articulation

of those consonants which require control of the air stream under pressure, as in p, b, d, t, k, v, f, s
2. Nasal alar constricting movements during continuous speech as an attempt to valve the air stream at the aperture of the nose
3. Inability to hiss, whistle, gargle, blow out candle or inflate balloon
4. Loss of fluid through the nose when drinking.

The following signs are pathognomonic of congenital submucous cleft palate:

1. Oral inspection reveals a relatively short palate and large oropharynx at rest and during phonation or gagging
2. Bifid uvula
3. Submucous defect in soft palate revealing lack of continuity of muscle across midline
4. Palpable notching in posterior border of hard palate indicating submucous osseous defect
5. Anterior displacement of palatal aponeurosis or velum is suggested by a forward position of levator activity.

Diagnostic Tests. One of the simplest and most useful laboratory tests available to physicians can be found in the lateral x-ray film of the head. The head should be carefully oriented to obtain a midsagittal view. One film is obtained at rest and a second film during the sustained phonation of the vowel *u* as in "boom." In the normal the soft palate elevates to contact the posterior pharyngeal wall (Fig. 68). In palatal pharyngeal insufficiency or incompetence such valving does not occur (Fig. 70).

Other laboratory tests have been designed to measure the effectiveness of palatopharyngeal valving by recording air pressures escaping through the nose during continuous speech or during the phonation of isolated speech sounds.

THE GROWTH FACTOR

Structure and function, as described previously, are not static in a growing youngster. Therefore examination of the anomaly must include the fourth dimension of time and the modifications brought about by the process of growth and development. Because of such natural modifications the ultimate nature of the defect at the age of 16 years may be quite different from that observed at birth or at six years of age. The data accumulated in the longitudinal study of such patients have already demonstrated individual patterns of change of the cleft and adjacent parts as a function of time. In terms of the width of the cleft, one of several critical measures determining operability, the processes of growth may produce circumstances more favorable to surgery. In one child significant narrowing of the width of the cleft, and thus operability, may be evident at one year of age, and in another

at two or three years or even later. Some children will show no change in the lateral dimensions of the cleft, and in a rare few the cleft will become wider. Surgical procedures on the palate must be related not only to the quantity and quality of tissues available at any given time, but also to the possibility that more favorable circumstances in terms of structure and function may develop at a later stage in the child's development (Fig. 72).

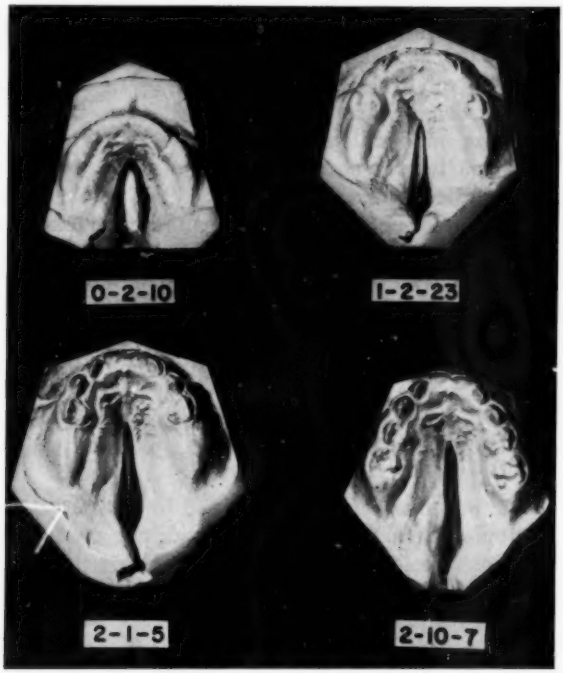

Fig. 72. Changes in the width of the cleft as a function of growth. Serial casts beginning at 2 months and 10 days of age and extending to 2 years, 10 months and 7 days of age on a child with an unoperated cleft of the hard and soft palates. Note the gradual narrowing of the cleft and the increase in size of the palatal processes.

Timing of all therapeutic procedures becomes an individual matter. Each child must be observed longitudinally so that prescription of treatment is based on a careful assessment of the assets and deficits and their pattern of changes with growth.

ASSOCIATED FACIAL DEFECTS INFLUENCING TREATMENT OF CLEFT LIP AND PALATE

The oral and facial defect has origin from the first branchial arch of the embryo. Since multiple congenital anomalies are not uncommon,

it is not unusual for other derivatives of this arch to be congenitally deformed. Microtia, atresia or stenosis of the external aural canal, facial fistulas, micrognathia or temporomandibular joint growth disturbances are not infrequently associated anomalies (Fig. 73). In the Pierre Robin Syndrome,[21] for instance, mandibular micrognathia and glossoptosis are associated with a cleft palate. If the mandible is small or asymmetrically arrested in its development, adequate opening of the mouth essential for exposure to palatal surgery may be a problem. If the external canals diminish hearing, speech results of palatal pro-

Fig. 73. Right unilateral microtia with associated deformity of the right temporomandibular joint leading to arrest of growth of the mandible on the affected side, deviation of the mandible toward the involved side, and limited range of mandibular movements.

cedures cannot be evaluated until hearing is improved. The success of habilitation is, of course, dependent on integrated treatment of all these defects.

Associated Defects in Other Organ Systems

Additional congenital anomalies may involve other organs. Significant additional anomalies are estimated to occur in about 10 to 25 per cent of children with clefts.[7, 16] This figure tends to be higher in medical centers where selectivity of referral for more serious situations is present. When intracranial, cardiovascular, renal, respiratory tract or gastrointestinal defects are present, the cleft of the lip and palate may have a lower priority in therapy. For these and other musculoskeletal problems of the child appropriate treatment of each has a sequence determined by careful examination and specific planning. The physician who cares for children appreciates also that a child born with a congenital anomaly has no insurance against prenatal or birth injuries, prematurity or neonatal hazards, or subsequent complications of dis-

ease or accidents. It is in this perspective of possible multiplicity of handicaps that management of the child with a cleft lip and/or cleft palate may require the utmost in professional skills.

TREATMENT CONSIDERATIONS—INFANCY THROUGH ADOLESCENCE

THE NEONATAL PERIOD AND EARLY INFANCY

The immediate concern after birth is with the usual extrauterine physiologic adjustments. The newborn with a cleft usually poses no special problem unless there are associated anomalies of vital organs or a small mandible with a tongue obstructing the airway. Otherwise the most urgent concern is to inform the parents that their baby is not perfect.

Family Reactions

The birth of a deformed baby presents an understandable emotional crisis to the parents. The high premium placed on physical perfection and especially on facial features makes this birth a catastrophic climax to months of hopeful expectation. An immediate shock reaction is predictable—the questions, denials, guilt feelings and pity for self and child are common experiences. It is at this time that the physician needs to establish his leadership in support, interpretation and guidance for the family. Counseling takes more than mere assurance that all will be well eventually, and requires a continuous program. Readiness of the parents to understand is geared to their early and subsequent emotional adjustment and to their maturity and stability as adults and parents. The early contributions that the family physician, obstetrician or pediatrician can make are manifold: (1) relieving guilt feelings of mother and father; (2) promoting an emotional climate through guidance which assists parents in accepting their child; (3) planning a treatment program which effectively utilizes specialized skills. The physician should be aware of family reactions which may superficially appear healthy, but which, on closer examination, reveal evidence of disturbance. Oversolicitude or an overrelaxed attitude in infant and child care may be a less readily recognized sign of maladjustment than overt rejection and neglect. Each may be penalizing to good physical and emotional growth of the child.

Interpretation is not limited to parents. Siblings, relatives, neighbors, community, school and, later, employers need in their own language to understand. In our society emphasis is placed on physical attractiveness and intelligible speech for vocational progress, marriage and success in

general. There are universal social attitudes that if an individual looks different and speaks differently, he must be different and thus becomes socially less acceptable.[1] It is in such a perspective that the habilitation process begins and finds some of its goals.

Feeding the Infant

The parents' needs have a high priority, but soon the infant requires attention. In spite of the deformity imposed by the cleft in the lip and palate, the feeding procedure may not be complicated. The infant readily adapts to his deficiencies in sucking and swallowing. Patience in feeding and minor adaptations of the nursing unit are the only adjustments usually necessary. Breast feeding, of course, is not likely. In the initial feedings after birth, or before and after lip surgery, a medicine dropper with a soft rubber tube extension can be used. Swallowing may cause loss of fluids through the nose, but this can be minimized by positioning the infant in a sitting posture on the mother's lap. The need for gavage feedings is infrequent.

During nipple feedings the infant's tongue strokes the nipple. When sucking is possible, the tongue-nipple manipulations are of little consequence. However, in the cleft palate such movements can be advantageous to replace the sucking loss. A softened, flexible nipple with openings slightly enlarged, preferably by a cross-cut in the inverted tip, will serve to make this tongue action more efficient. Air swallowing is more common, and frequent burping is in order. Excoriation and infection of a friable lip is a danger and should be avoided lest operation be delayed. Since the infant with a cleft is vulnerable to aspiration, avoiding large volumes of milk in the infant's mouth and unhurried feedings will minimize this danger.

In infants with extremely wide clefts or with small mandibles and retroposed tongues a "lamb's nipple" may be used to advantage. The high degree of success in using modified standard approaches to feeding these infants has made obsolete any need for a special plastic appliance to cover the cleft during feedings. Even rubber palatal flaps, as in the Brophy cleft palate nipple, have been found unnecessary, if not ineffective. For the parents' sake it is probably advisable not to complicate the feeding experience with different nipples and procedures which further emphasize the difference of their baby.

Difficulties of feeding are less often due to the cleft lip or palate than to improper techniques or the presence of associated defects such as cerebral or metabolic disorders, respiratory obstruction or other congenital anomalies of the mouth and gastrointestinal tract.

Cleft Lip Surgery

Cleft lip surgery is an early infancy procedure, while palatal operations are done later in infancy or during early childhood, if possible. The timing for lip surgery varies with the habits of the surgeon, but also with the extent of the defect, the health status of the infant and sometimes with the adjustment of the parents. The usual procedure is to wait for progressive infant weight gain. Weight under 7 pounds, the presence of lip tissue friability or a wide bilateral cleft may cause further delay. An infant with a malformed ear may cause unexpected problems in endotracheal intubation because of the frequently associated temporomandibular joint anomaly which restricts mouth opening.

Preoperative care is no different from that for other elective surgical procedures in infancy. Adequate nutritional status, normal hydration and absence of infection are essential. Lip surgery is associated with blood loss, and, in contrast to older children and adults, even small amounts may be critical in the face of the infant's smaller total blood volume. Preoperative venoclysis, scalp vein or cut down, and available carefully matched blood are necessities. Preanesthetic medication requires careful consideration, since standard doses based on empirical formulas are frequently too large.

Postoperative care requires knowledge of the surgical and anesthetic chain of events. Anesthetic procedures are problems, since the surgical zone and sterile draping of the face and head make difficult the essential vital observations. The technical skills required, the need to control depth of anesthesia, oxygenation and respiratory exchange, and to remain out of the surgeon's way and yet safeguard the patient require the services of an anesthesiologist who is skilled in handling small infants and experienced with cleft lip procedures.

The details of operative technique vary with the training and experience of the plastic surgeon. The underlying objectives common to all procedures are to unite the cleft edges and to provide a mobile, cosmetically attractive lip. Inadequate results consist of an abnormally short, long, tight or badly scarred lip and notching of the vermilion border. An oronasal fistula in the anterior sulcus is also a common complication, but less important cosmetically and probably functionally. Deformities in the nasal cartilage may also persist after repair of the lip. Operations to correct such undesirable results are often necessary and are generally planned in the later preschool age period. In the case of a nasal deformity further surgery may be delayed until early adolescence.

Changes Incident to Lip Surgery. The immediate changes that follow repair of the cleft lip are not necessarily indicative of the ulti-

mate appearance of the lip, nose and facial profile. It must be remembered that the child is growing and that the changes incident to growth may lead to further improvement in facial appearance. On the other hand, surgical procedures which involve excessive manipulation of tissues for the sake of producing the most complete cosmetic result early may resolve into a deformity at some later age.

Surgery of the bilateral lip deserves additional comment. It is frequently a difficult procedure and is often performed, by choice or necessity, in two stages. A complicating feature may be the overlong or displaced vomer which causes the central prolabial process to protrude beyond the tip of the nose. The columella is frequently deficient, and as a result the repaired lip will yield a flattened and depressed nasal tip. Ultimately the differential processes of facial growth serve to minimize much of this deformity.

LATE INFANCY AND EARLY CHILDHOOD

Considerations for Palatal Surgery

In contrast to the greater uniformity of opinion regarding lip surgery, there is considerable controversy as to the indications, the timing and the procedures to be done in palatal surgery.

It appears, however, that most modern surgeons are concerned with three objectives: anatomic closure, normal speech, and avoidance of injury to growing structures. The divergence of opinion among surgeons seems to stem from failure to define criteria for readiness for palatal surgery and lack of agreement as to which surgical procedures constitute a hazard to the growth of the structures involved. It is also noted that standardized methods for estimating success, in terms of speech and facial growth and development, have not been uniformly applied in the judgment of surgical results. Moreover, reports of patients operated upon often do not present analyses of the original deformity, and thus the efficacy of the surgical effort cannot be evaluated in relation to the severity of the defect. These difficulties can be resolved by controlled investigation and agreement among the medical, dental and educational specialists as to proper measures for successful habilitation.

At the Cleft Palate Center of the University of Illinois infants with clefts have been observed for a sufficient length of time to allow certain preliminary judgments on the results of operation. It is clear that mere restoration of anatomical continuity across the cleft is not sufficient to assure functional success in all cases. The explanation for the variable results in the hands of the same surgeon using identical procedures resides mainly in the fact that there is considerable variation in the severity of the original deformity.

Preoperative appraisals based on the quantity and quality of tissues adjoining the cleft, the width of the cleft, the length of the soft palate, the configuration of the nasopharynx and the pattern of palatopharyngeal muscle activity indicated that some children are obviously better candidates for palatal surgery than are others. The differences of opinion that persisted after such presurgical analyses had to do with value judgments as to what constituted adequacy of tissue or length of soft palate, and so forth. There is good reason to believe that with follow-up studies, using the exact measures of plaster casts, cinefluoroscopy, cephalometric roentgenography, and laminagraphy, closer agreement on value judgments will be reached.

Timing of Palatal Surgery

The experiences gained so far in these longitudinal studies indicate that there is no one optimal age for repair of the cleft palate.

Timing is an individual matter and is related to the anatomical and functional assets present in the patient. Preoperative screening of patients, according to the criteria described above, assures a greater degree of functional success than does random selection without recourse to such analysis.

Early palatal surgery does not jeopardize the growth of tissues, provided (1) proper circumstances exist for conservative methods of palatal repair, and (2) provided such surgical methods are utilized. Conservative palatal surgery is defined as that which does not use relaxing incisions, extensive undermining and displacement of mucoperiosteum, fracture of bone or destruction of blood supply.

Early repair of the cleft palate is advocated in many circles on the grounds that such reconstruction facilitates the development of normal speech and avoids the perverted patterns of speech that require prolonged re-education at some later age. To counter this objection to delayed surgery, children with unoperated clefts are fitted with prosthetic speech appliances designed to obturate the cleft in the hard palate and to assist the muscles in nasopharyngeal valving.

Speech appliances are fitted as soon as the deciduous teeth are fully erupted and the child exhibits a level of emotional and social development sufficient to accept the prosthesis. These appliances have been fitted for children as early as two and a half years of age (Fig. 74). With the prosthesis, speech intelligibility often proceeds normally and sometimes without benefit of formal speech training. It has also been observed that the use of these appliances provokes increased mobility in the palatopharyngeal musculature. In this sense the appliance may serve as a kinesthetic device stimulating the muscles into greater activity.

Many of these appliances are interim devices, since surgery is elected at a later age. Modified appliances are also used as interim devices when the palate is repaired in two or more stages. By combining surgery and prosthesis, the child is not handicapped by the lack of anatomical structures essential to the control of the air stream and to the development of normal speech. In other children, when the deformity is so severe as

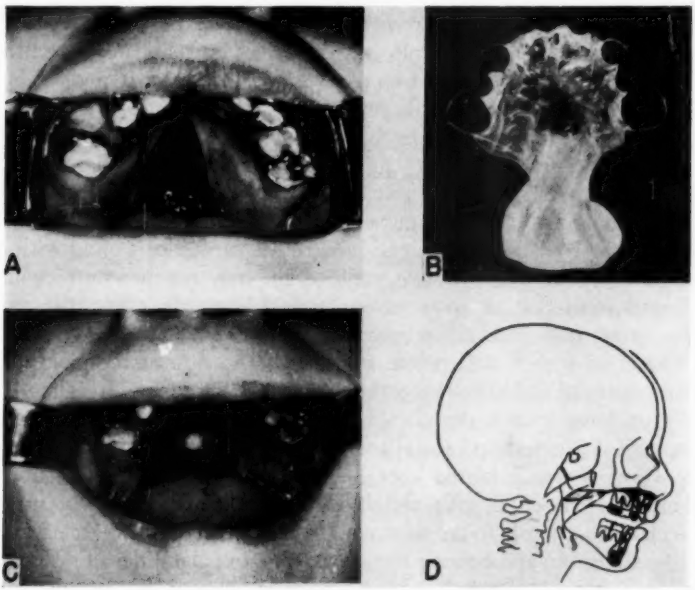

Fig. 74. Speech appliance for a child of preschool age with an inoperable cleft of the hard and soft palates. *A*, Note 4 orthodontic bands placed on the teeth to assist in the retention of the appliance. *B*, The plastic appliance with 4 clasps. *C*, The appliance in position. *D*, Tracing of the lateral cephalometric film reveals the stippled pharyngeal section of the appliance properly positioned to assist in palatopharyngeal valving.

to rule out surgery, the prosthetic speech appliances are modified from time to time in accord with the growth of the child and become the permanent means of speech habilitation.

THE SPEECH DEFECTS OF CLEFT PALATE

Cleft palate cannot remain a hidden anomaly. The defect is made conspicuous by the marked speech disturbance. Such speech is characterized by the abnormal escape of air through the nose and is associated with misarticulations and an excessive nasal quality to the spoken words. Since intelligible speech is one of the most distinguishing

characteristics of the human being, it is no overstatement that, to the person with a cleft palate, the speech defect is the single most handicapping aspect of his deformity.

Most English spoken sounds require a controlled oral emission of air with the exceptions of the consonants *m, n, ng,* which are largely nasal sounds. All sounds require a careful posturing of the tongue in relation to the teeth and palate in association with varying intraoral air pressure gradients.[14] In cleft palate speech the vowels are nasalized, and consonant sounds such as the plosives, fricatives and sibilants are either omitted, distorted or replaced by other sounds more easily made.

Significantly, the speech defect of cleft palate, and other entities producing palatopharyngeal incompetence, is not always readily detected in infancy and early childhood. The neuromuscular patterns characterizing early speech development do not call for refinements of oral air control, tongue movements and palatopharyngeal activity essential for later normal speech. Indeed, the speech of a child with an unoperated cleft palate at two years of age may not be conspicuously different from that of some of his normal peers. In effect, the child often grows into cleft palate speech.

The child with a cleft palate is also liable to other common speech disturbances of children. It is estimated that 5 per cent of all school age children have speech disorders of such severity as to interfere with educational progress and social and emotional development.[2, 12] Under five years of age infantile speech patterns are exceedingly frequent. Commonly the child with a cleft palate has an admixture of speech defects due variously to developmental language immaturity, malformed dental structures and hearing loss in addition to palatal defect. Whether the child is encouraged or guided wisely, or rebuffed and mismanaged, as he reaches out to communicate with those around him is an additional consideration. The attitude toward speech may either provide proper motivation or set limitations to intelligible speech development. Thus factors other than those related directly to the cleft may need attention during the language development of the child.

After operation or the placement of a prosthetic speech appliance the child does not automatically benefit in terms of improved speech. Speech therapy is needed in most cases as a significant follow-through. Since speech development normally extends through the first eight years of life, it is to be expected that the procedures necessary for correcting faulty habits and developing maximal benefit from new structures may require an equally extended program of speech training. Such training brings to fruition the many services for the child with a cleft palate and should be considered an integral aspect of the habilitative program.

CHILDHOOD THROUGH ADOLESCENCE

Medical and Dental Considerations

Surgery is only a single milestone for many children with cleft lip and cleft palate. New problems become apparent as the teeth erupt, speech develops, infections and emotional experiences accumulate for the child.

Hearing loss is a special threat. Chronic and recurrent otitis media is a common medical problem in these children, requiring frequent careful evaluation and treatment. Audiometric studies have indicated that 25 to 90 per cent of children with cleft palate have otologic changes and evidence of hearing loss.[4, 26] At the Cleft Palate Center 40 per cent of all children with palatal disturbance had a hearing loss in one or both ears of at least 15 decibels in many speech frequency ranges. Comparatively, hearing loss is found in about 5 per cent of the general school population.[15] The inability to discriminate sounds in speech and the loss of self-monitoring make moderate or severe hearing loss catastrophic to a child with cleft palate speech. Amplification may need to be considered if the hearing threshold for speech is below 35 decibels in both ears.

Tonsils and adenoids may perform a critical function for intelligible speech in a child with an operated cleft palate.[18, 20, 29] In successfully repaired cleft palate, in submucous cleft palate or other potential palatopharyngeal inadequacy an ill-considered adenoidectomy may precipitate a severe and permanent speech disturbance.

The mass of adenoid tissue in these circumstances subserves a function for normal palatopharyngeal closure. In the event that the palate is deficient in length or mobility, or if the lateral pharyngeal muscles are comparatively inactive, the adenoid mass may minimize the latent inability to produce nasopharyngeal valving by its static protrusion into the epipharynx. The adenoid is less important to palatopharyngeal function in the adult because of its negligible size and, more significantly, because palatopharyngeal closure obtains at a level below the site of the adenoid. In circumstances of dependence on the adenoid for palatopharyngeal closure its gradual involution may be less disturbing than its sudden removal. The slower process probably allows for the development of compensatory muscle activity.

The role of the tonsils is less well defined, since their relation to the faucial pillars and their posture within the pharynx are so variable. In some instances markedly hypertrophic tonsils may distort the nasal-oral balance of the air stream during speech or, by their size, impede the full range of elevation to the soft palate. In such instances, or if chronic infection is contributing to Waldeyer's ring pathology, tonsillectomy without adenoidectomy may be indicated. In other instances the tonsils

may actually contribute to palatopharyngeal closure, and in such event they warrant due consideration and preservation.

All intraoral surgical procedures in these children should be the responsibility of a plastic or otolaryngologic surgeon familiar with the specific problems of a child with a cleft palate. Appropriate consultation with a speech pathologist may be warranted if there is any doubt as to the proper role of the tonsils and adenoids in speech.

In the event that chronic otitis media, moderate or severe hearing loss requires consideration of an adenoidectomy, a careful review of medical and conservative surgical procedures is indicated. The possibility that adenoidectomy may prevent further hearing loss needs to be weighed against the probability of serious speech deterioration. Consideration of further intensive otologic treatment and local and systemic antibiotic therapy is always warranted. In select instances continuous prophylactic antibiotic therapy has been found helpful in minimizing recurrent pharyngitis and otitis media. If operative methods need to be used, local radium therapy, tonsillectomy alone or a partial, lateral, direct-view adenoidectomy should be considered before removing the entire adenoidal mass. Midsagittal lateral x-ray films of the head taken in a rest position and during the phonation of *u* will frequently indicate the degree of dependence upon the mass of the adenoid during palatopharyngeal closure.

Dental health requires continuous vigilance, as does the preservation of hearing. The physician is in a unique position to make early referrals of his child-patients to a dentist willing and skilled to work with children. At two to two and one-half years dental cavities may already require attention, and a program of dietary sugar control, topical fluoride application and instruction of parent and child in oral hygiene may need to be instituted. Frequent dental examinations are often necessary. The preservation and care of the deciduous dentition is especially important to the child with a cleft palate. Without these teeth articulate speech is impeded, orthodontic treatment is impossible, and the retention of dental prosthetic appliances, when required, is complicated. Speech therapists with experience in the problems of cleft palate consider good dental health one of the child's most important assets to intelligible speech.

Orthodontic treatment for the cleft palate child may begin with the deciduous dentition. The purpose of such treatment is to realign the palatal segments in order to obtain a more normal contour to the alveolar ridge, reshape the palatal vault, and provide a more symmetrical foundation for the support of the lips and nose.[19, 27] Such changes are possible because orthodontic treatment in cleft palate allows for the movement of palatal segments in addition to altering the position of

the teeth within the bone (Fig. 75). Because the roof of the mouth is also the floor of the nose, realignment of the palatal processes not only produces desirable alteration in the contour of the palate, but also induces similar changes within the floor of the nose (Fig. 76).

Success in orthodontic treatment is largely dependent on the antecedent surgical procedures. Excessive cicatricial tissue and tight binding labial musculature restrict the growth of the maxilla and resist orthodontic expansion.

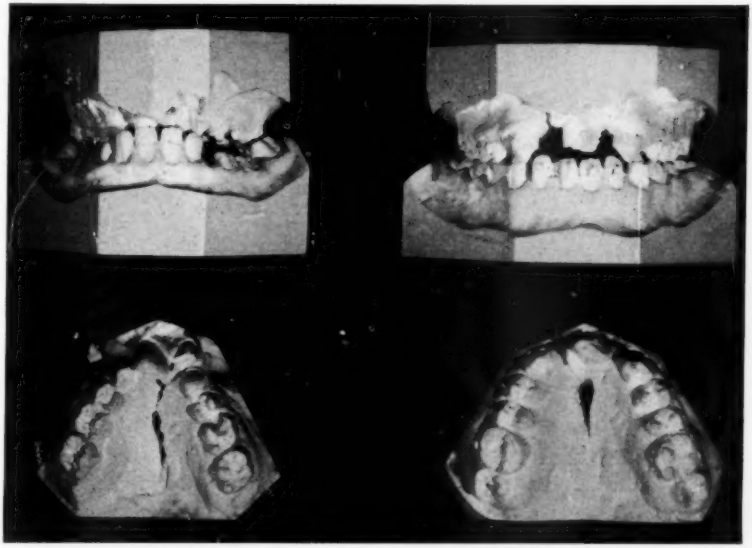

Fig. 75. The changes incident to orthodontic treatment of a repaired bilateral cleft lip and palate. The articulated casts on the right indicate the improved occlusion following treatment. The maxillary casts below indicate the degree of expansion obtained by lateral movement of palatal segments.

Orthodontic treatment may extend over a number of years, beginning about four years of age, and is not complete until the permanent dentition is in good occlusion. Two or more periods of active treatment, with observation and guidance in the interim, are generally required.

Prosthetic dental care is concerned with the replacement of missing or malformed teeth in the line of the cleft and, when indicated, with the design of speech appliances.

In the event that operation is contraindicated or delayed or, if performed, does not produce proper palatopharyngeal closure, a dental speech appliance may substitute satisfactorily. Such appliances may be

made of acrylic plastic retained by clasps about the teeth. Metallic castings of nonprecious and precious metals may also be used in the design of speech appliances.

For purposes of description, the appliance consists of three parts: the palatal, the velar, and the pharyngeal sections. The palatal section may consist of little more than a covering for the hard palate and carry the clasps for attachment to the teeth. This section may also supply replacements for missing teeth. In cases in which vertical dimension must

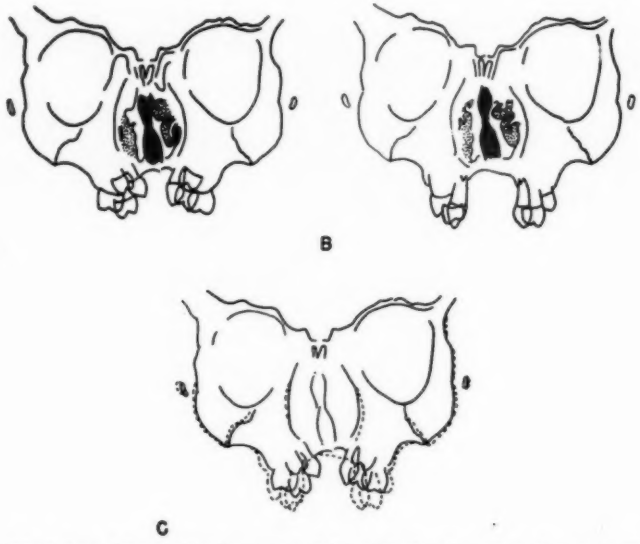

Fig. 76. Oriented frontal laminagrams of the orthodontic patient shown in Figure 74. The superimposed tracings reveal the altered position of teeth within the bones as well as the extent of lateral movement of the maxillary bones with concomitant increase in the width of the nasal chambers.

be increased because of arrest of vertical growth of the middle face, the palatal section may cover the natural teeth. In such circumstances the teeth should be crowned in gold to protect them from dental caries. By forward extension the palatal section may also plump the upper lip and improve the facial profile.

The velar section is in the shape of a bar connecting the palatal section to the pharyngeal portion of the appliance. It is the pharyngeal portion that extends into the nasopharynx and assists in palatopharyngeal closure (Fig. 77). The skill of prosthodontists in designing these devices has added a fortunate dimension to speech habilitation of cleft palate whenever surgery is unable to provide such results.

EMOTIONAL HEALTH CONSIDERATIONS FOR THE CHILD WITH A CLEFT

In the past decade of child health practices increasing emphasis has been placed on healthy personality development.[30] For the handicapped child, no less than for the well child, it is a positive health goal requiring the physician's awareness and special attention. The child with a cleft lip and/or palate requires informed insights. From the onset of birth the parent-child relationship merits active surveillance. An accepted, loved and secure child should be the central figure in the treatment program. In spite of our highly specialized skills, a cosmetically

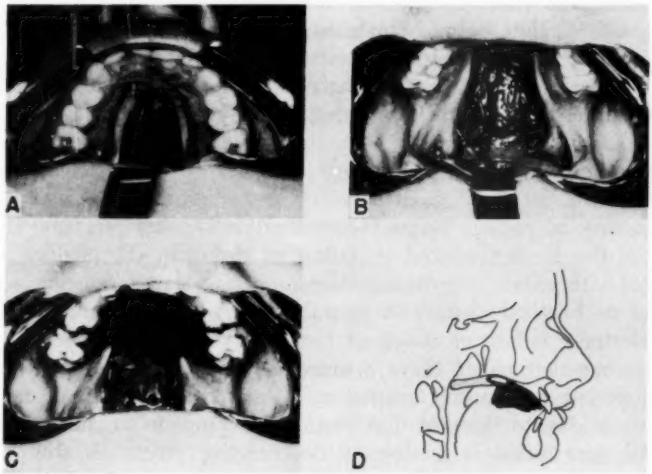

Fig. 77. Prosthetic speech appliance for young adult with inoperable cleft of the hard and soft palates. *A* and *B*, Revealing the extent of the deformity. *C*, Intraoral view of the appliance. The palatal section is fabricated of a nonprecious metal, and the pharyngeal section is of clear plastic material. *D*, Tracing of a lateral cephalometric roentgenogram revealing the appliance in position. The stippled extension indicates the position of the pharyngeal section at the critical point of nasopharyngeal valving.

attractive face and intelligible speech are not always early or assured accomplishments. Many surgical, medical and dental therapies may be necessary, and the child's living experiences are sometimes complicated by a community of children and adults reacting with variable hostility or maudlin sympathy.

Fortifying the child in the home with understanding parents and supporting him in the various traumatic exposures are essential. Preparation for hospitalization and for the variety of medical, dental and educational procedures merits considerable time and effort to minimize the fears and anxieties of both patient and parent. The emotional

problems encountered can be anticipated with knowledge of the child's basic endowment, parent-child adjustment, and preparation given for daily living. In general, appropriate guidance requires an awareness of potential hazards and an appreciation of the climate all children need for healthy living.

FAMILY COUNSELING

Etiological Factors and Eugenic Aspects

Parents, and later the affected child, will invariably want to know the causes of cleft lip and palate and the possibility of recurrence in their family. The exact answer is not known. Some generalizations are available from embryologic, teratogenic and genetic studies, and with careful interpretation such information may be a basis for family counseling.

The embryologic chain of events indicates that the defects occur between the fifth and twelfth weeks of gestation. Significantly, the defect may be present before the mother realizes her pregnancy. The cleft of the lip is produced by failure of fusion of the median nasal process with either or both maxillary processes, thus producing a unilateral or bilateral cleft. The palatal cleft is a later occurrence and constitutes a failure of union of the lateral palatine processes. In a submucous cleft palate there is mucosal union, but incomplete bone development and muscle coaptation. Whether this defective organogenesis is due to arrested development, incomplete or absent mesodermal penetration or perhaps a degenerative process is still to be resolved.

Animal studies have broadened our knowledge. A variety of teratogenic agents are able to produce multiple congenital anomalies, including cleft palate, in specially inbred animals. These studies have suggested that a specific noxious agent is less important in producing anomalies than the susceptibility of certain embryonic tissues at the time at which the agent was applied. On the basis of available information Fraser and others[9] have generalized: "Congenital defects, except for those showing regular Mendelian inheritance, cannot be attributed wholly to genetic or wholly to environmental causes but are the result of the interaction between the two" and possibly also to a "factor of intrauterine variability that causes either differences in the amount of teratogen reaching the fetus or differences in the fetal reaction to it."

Genetic studies of families with affected children have suggested that cleft lip with or without cleft palate should be considered separate from cleft palate alone. Fogh-Andersen[7] in Denmark found a family

incidence of 27 per cent for cleft lip alone, 41 per cent for cleft lip and palate, but only 19 per cent for isolated cleft palate. Sanders[22] in Rotterdam found that 44.5 per cent of all cleft patients studied had a positive family history. In this country a lower incidence of 20 to 30 per cent is frequently reported, explained somewhat on the basis of relatively smaller families and less complete records as compared with the European figures.

Although medical genetics is still in an early stage of its scientific development and exact information is not available, certain "risk rates," empirically determined, can be used as a background for family counseling. The studies of Fogh-Andersen[7] provide a less threatening prognostication than the generalization of Murphy[17] that the likelihood of subsequent children with anomalies is approximately 24 times greater than for parents with normal children. Fraser,[8] using Fogh-Andersen's data, summarized the possible recurrence in a family with an affected child or parent:

"Risk-Rate" for Cleft Lip (with or without cleft palate)	
For siblings—one child affected and parents normal	4%
For siblings—one child affected, one parent affected	14%
For offspring—one parent affected	2%
"Risk-Rate" for Cleft Palate (without cleft lip)	
For siblings—one child affected and parents normal	2%
For siblings—one child affected, one parent affected	17%
For offspring—one parent affected	7%

Experimental studies, theories and statistical data, of course, do not often help the bewildered family. If not properly interpreted, such information may threaten and disturb the marriage and parent-child relationship rather than inform and guide in family planning. The eventual decision about future children may depend upon the experiences and outcome of treatment with the affected child. Such counseling remains, however, an important consideration for the physician in his family practice.

SOCIO-ECONOMIC AND COMMUNITY ASPECTS

Economic and professional manpower resources, as well as special knowledge and highly developed technical skills, are required in a comprehensive treatment program for children with cleft lip and cleft palate. Other handicapping conditions among children share similar requirements. Few families and communities are equipped to provide all the support necessary for such major habilitative efforts. Wherever possible, medical-dental centers and community resources usually need to integrate to provide the essential treatment.

For cleft lip and cleft palate the diagnostic, treatment-planning and

surgical phases are important, although among the less time-consuming events. The dental, otologic and speech training aspects require recognition as long-term activities. In contradistinction to the relatively brief hospitalization periods, general dental and prosthetic care involves continuous supervision and orthodontic therapy entails years of treatment and observation. Speech training may be necessary for many of the child's preschool and school years. Otologic and audiologic surveillance adds still another dimension to the long-term management program.

The need for such specialized resources, and often for financial support as well, makes cleft lip and palate a larger community problem. It is common for the majority of such patients to be referred to the state services for crippled children. In Illinois the Division of Services for Crippled Children is an integral part of the University of Illinois and is intimately related as a member of the Cleft Palate Center team. As a medical care agency it provides services in many ways for a child with a cleft. Case findings, initial evaluation in community clinics and referral to specific areas for treatment are a preliminary activity in cooperation with the family physician. An outstanding contribution is the integration of the community and state-wide resources in conjunction with the medical center program. Financial support is extended if necessary to provide for specialized therapy. Follow-up care, as a necessary adjunct to any habilitative program, is usually maintained until 21 years of age unless treatment can be successfully terminated earlier.

All states have a crippled children's program which provides for medical and related care services for handicapped children. There are, however, many differences among such programs in the 48 states. Among the differences are the medical categories which may receive services. Probably all give total or partial care to children with cleft lip and palate. The recognition of the need for such support is obvious in the socio-economic consideration of optimum care for most handicapped children.

CONCLUSIONS

Cleft lip and cleft palate offer a degree of habilitativeness that is not shared by many other equally serious congenital or acquired handicaps. To achieve this potential requires careful planning and skillful execution of treatment which respects the child's growth, development and individual variations.

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1853 West Polk Street
Chicago 12, Illinois

CLEFT LIP AND CLEFT PALATE

Surgical Considerations

WAYNE B. SLAUGHTER, M.D., D.D.S.

SAMUEL PRUZANSKY, D.D.S.

HAROLD L. HARRIS, M.D.

The aims of cleft lip repair are to produce a near-normal lip which is functionally and cosmetically acceptable; to correct the deformity of the external nose, nasal cavity and alveolar process, and thereby establish an environment conducive to normal growth and development of the face and mouth structures.

The primary goal of palatal surgery is to provide a mechanism requisite to the establishment of normal speech. Related to this primary objective is the necessity of establishing as normal a form to the palate and dental arches as possible; to reposition the segments by physiological means in order to construct a soft palate of adequate length and mobility for functional occlusion of the nasopharyngeal passage.[7]

Surgery for cleft lip and palate demands specialized training and experience in such procedures. In addition, the surgeon must have a full understanding of the nonsurgical problems essential to the program of complete treatment and be prepared to cooperate with specialists in allied fields. These special problems are inseparable from his immediate surgical responsibilities.

From the Children's Center, St. Francis Hospital, Evanston, Illinois.

ANESTHESIA AND SUPPORTIVE CARE

The minimal morbidity and negligible mortality incident to surgical repair of lips and palates are due largely to careful anesthetic management and consideration of principles of nutritional and supportive care. A primary problem during anesthesia is maintaining an unobstructed airway during the period of surgical manipulation. Blood and mucus collecting in the airway constitute a principal hazard. The immaturity of the patients, the possibility of dehydration, and bleeding leading to shock and death emphasize the need for scrupulous attention to nutritional and supportive management.

Blood Replacement

With careful hemostasis minimal bleeding in a primary cleft lip repair will be at least 50 to 75 ml. (16 to 25 per cent of the blood volume of an 8 pound baby). This seemingly minimal bleeding is relatively massive. Such blood loss, coupled with the vasodepression of anesthesia and superimposed upon a background of malnutrition, dehydration or anemia, can quickly lead to a circulatory crisis. Bleeding is frequently greater than the minimum and must be recognized and corrective measures instituted immediately.

Blood replacement is calculated to be volume for volume replacement of that lost at operation plus an amount that will raise the postoperative hemoglobin above 12 gm. per 100 ml. if preoperative anemia was present. Blood volume is estimated to be approximately 45 ml. per pound of body weight in small infants. This value gradually reduces to about 40 ml. per pound of body weight at age two years. Thus, if the baby weighs 10 pounds (theoretical blood volume 450 ml.), has lost 100 ml. of blood at operation, and had a preoperative hemoglobin of 10 gm. per 100 ml., he will require approximately 180 ml. of transfusion to raise his postoperative hemoglobin to 12 gm. per 100 ml. This relatively large amount of blood (42 per cent of theoretical blood volume) should be administered during and immediately after operation to prevent shock and to avoid the overload of the circulatory system by too rapid infusion.

If transfusion is delayed until the first postoperative day, the risk of development of profound shock is increased. In the event that blood volume has re-expanded because of hydration and osmotic homeostasis, the risk of circulatory overload again becomes a probability and requires due caution.

The so-called physiological anemia (hemoglobin values of 10 to 11 gm. per 100 ml.) in infants is a manifestation of the net difference in the rate of body growth and the rate of elaboration of red blood cells

and hemoglobin. One hundred milliliters of blood lost by a 15 pound infant (blood volume 650 ml.) with a preoperative hemoglobin of 10 gm. will leave the postoperative hemoglobin at approximately 8.5 gm. Anemia of this degree diminishes the oxygen-carrying capacity of the blood to the point at which the probability of successfully combating stress situations and healing of suture lines is diminished. If the operation is the first of a series, the anemia will increase the hazard of succeeding anesthetics and operations. Thus it seems logical to transfuse to optimal hemoglobin levels at the time of the first procedure, rather than wait until a critical situation is created. In all instances anemias should be investigated for cause and treated preoperatively, if necessary.

Preoperative hemoglobin values as low as 8 gm. may be encountered in infants who were premature. This is preferably corrected preoperatively. Transfusion of older children (two to three years old) who are submitted to cleft palate surgery follows similar principles of volume for volume replacement of blood lost at operation plus an amount that will raise the hemoglobin above 12 gm. per 100 ml. These are general principles and are applicable to children who are operated on for other conditions.

Fluid Replacement

An infant is not considered to be ready for elective surgery and the attendant anesthesia until he has regained birthweight, is eating and gaining regularly, and the hemoglobin is above 10 gm.

On the operative day oral foods and fluids are withheld for six to eight hours before operation. Infants receive 150 cc. of 5 per cent glucose in water parenterally about two hours before operation. Feeding is resumed after operation as tolerated by the baby. Twenty-four hours postoperatively the infants are usually back on regular feedings. Parenteral fluids are given to supplement the oral intake. Oral plus parenteral fluids on the operative day should total 75 to 100 cc. per pound of weight. During the heat of the summer the higher value is used. Children two to three years old who have undergone palatal surgery may not eat or drink well for 48 hours after operation, and their intake may have to be supplemented by parenteral feeding.

Failure to maintain the state of hydration and caloric intake will lead to metabolic acidosis, hyperpyrexia and even convulsions. Respiratory acidosis superimposed upon a metabolic acidosis is the background for convulsions during anesthesia. The cerebral edema following such an episode can be fatal. Fever in the immediate postoperative period following aseptic surgery and uncomplicated anesthesia is most commonly due to dehydration.

Anesthetic Procedure

The administration of anesthetics to cleft lip and palate children is best managed by anesthesiologists who have had broad experience in the use of inhalation drugs in pediatric patients. They should also have a background that will facilitate their understanding of the anatomical problems inherent in endotracheal intubation of patients with abnormal air passages.

Inhalation drugs introduced through an endotracheal tube are preferred in both the cleft lip and cleft palate group. Inhalation drugs (diethyl ether, cyclopropane and nitrous oxide) can be used with an abundance of oxygen to provide the necessary relaxation, and are readily eliminated so that the child will return to consciousness at the completion of the operative procedure.

Use of narcotics and other basal sedatives is best avoided in small infants. Prior to lip repair infants are premedicated with atropine or scopolamine in minimal dose sufficient to suppress secretions. Children 18 months to three years of age are given codeine (15 to 30 mg.) with the scopolamine as a mild sedative prior to anesthesia for repair of the palate. Heavier sedation has been found unsatisfactory because the accompanying respiratory depression retards induction of anesthesia and intubation; respiration is sluggish during the anesthetic period, and the recovery is delayed. If the child fails to awaken about the time the endotracheal tube is removed, he is in immediate danger of respiratory obstruction from vocal cord spasm or aspiration of secretions or vomitus. Prolonged sleep will also delay his return to feeding and aggravate the complications due to dehydration.

The immaturity of the respiratory mechanism in infants under four months renders them especially susceptible to the development of hypoxia. Drug-induced depression of the respiratory center will allow such patients to slip from breath holding, vocal cord spasm or other respiratory obstruction to asphyxial arrest as a rapidly progressive transition. Hence the insistence upon the necessity of the child's being awake when the anesthetic is discontinued. Care must be taken to remove all mucus, blood and clots from the pharynx before extubation. Even though the infant is awake, he must be watched closely after repair of a wide cleft of the lip. Prior to closure of the lip defect the infant breathed at least in part through the cleft. After the closure it may take a few hours for him to learn to keep his lips separated. A few moments of obstruction from closed lips may induce a fatal asphyxial episode if unattended. Such lips may have to be mechanically separated for a few hours by the application of traction to the lower lip.

Endotracheal intubation of patients is preferred. The tube will guard the lower respiratory tract from contamination with blood and mucus,

will provide for maintenance of smooth anesthesia with continuous oxygenation, and will allow the anesthesiologist to conduct his affairs without directly entering the surgical field. Orotracheal intubation for cleft lip and nasotracheal intubation for cleft palate surgery offers the minimum of interference in the surgical field.

The primary objections to the use of the endotracheal tube stem from faulty technique. Atraumatic introduction of a tube of proper diameter and length for the individual patient and the use of connectors that will allow the tube to be fastened so that it will not ride up and down in the glottis, kink, or slip down to occlude a bronchus is almost devoid of complications.

The protruding premaxilla and the relative shortening of the mandible are impediments to laryngoscopy and intubation. Intelligent survey and understanding of the significance of the deformities preparatory to precise and proper use of the laryngoscope will facilitate atraumatic exposure of the glottis.

The combination of local anesthesia with general anesthesia does not reduce the anesthetic requirements. The addition of epinephrine to the injected solution has not been found to significantly reduce the blood loss, and the tachycardia so induced has at times been alarming.

Appropriate antibiotics are administered only when infection develops or prophylactically to minimize the development of otitis media or other local or generalized infections. Sedation with phenobarbital may be required in children if restlessness is present. Enemas have not been found necessary as either a preoperative or postoperative procedure.

CLEFT LIP

Cleft lip is an anomaly that involves not only the lip, but also the external nose, the nasal cartilages, the nasal septum and the alveolar process. The more complete the cleft in the lip, the greater the derangement in these related structures. This constellation of deformities extending beyond the lip is not unexpected in consideration of the common anlage of these structures and the effects of a cleft lip upon their growth. The anatomy of the defect can be better understood from an analysis of the muscular imbalances that characterize the deformity.

Muscle Imbalance

In the description of the longitudinal changes in the face of the child with a cleft lip it is misleading to date the cleft from the time of birth. At birth the cleft deformity has been present for a significant

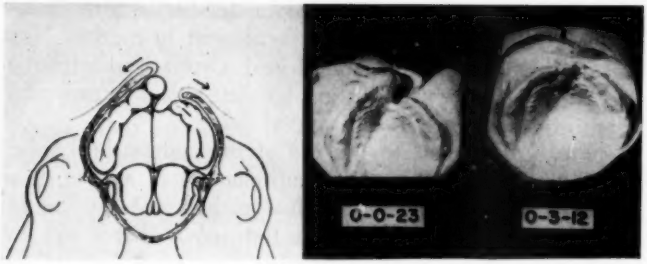

Fig. 78.

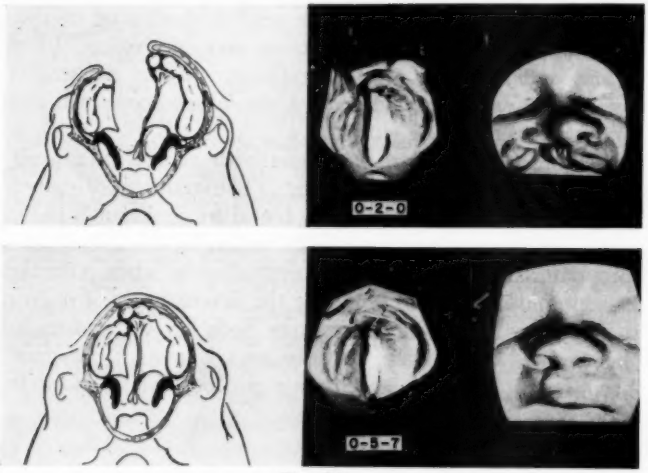

Fig. 79.

Fig. 78. Complete cleft of lip and alveolar process on left side before and after lip repair. Schematic diagram illustrates direction of muscle pull on segments. Plaster reproduction of the palate at 23 days of age was obtained prior to lip surgery. The changes recorded at 3 months and 12 days of age reflect the effects of lip repair in molding the segments into place.

Fig. 79. Complete unilateral cleft of lip and palate on right side, before and after lip repair. Upper series of illustrations indicate the lateral pull of the cleft lip on the palatal segments. The changes due to lip surgery are revealed in the lower series of illustration.

period of the infant's intrauterine lifetime. During this time exceedingly rapid fetal growth has taken place and the structures of the face and mouth developed without the normal enveloping restraints of the muscles of the lip and without the continuity of an intact alveolar ridge and palate. Particularly, it is the breech in the ring of muscle complex comprised of the orbicularis oris, buccinator and superior constrictor of the pharynx that accounts for the displacement of tissues and for the distortion in the growth of the parts involved (Figs. 78,

79). When there is also a cleft in the palate, the force of the tongue, as it plunges into the cleft space, contributes further to the lateral displacement of palatal segments.

Surgical reconstruction of muscle balance and re-establishment of normal antagonistic forces across the midline is the key to the establishment of an environment conducive to the proper growth of the affected organs. The problem is to establish this muscle balance without disturbing the growth potential of the tissues being manipulated and avoiding scars that will tie or bind down the normally expansive forces of growth.

Lip Repair

Repair of the defect in the lip entails an approximation of the skin, mucous membrane and muscular elements in accord with their proper

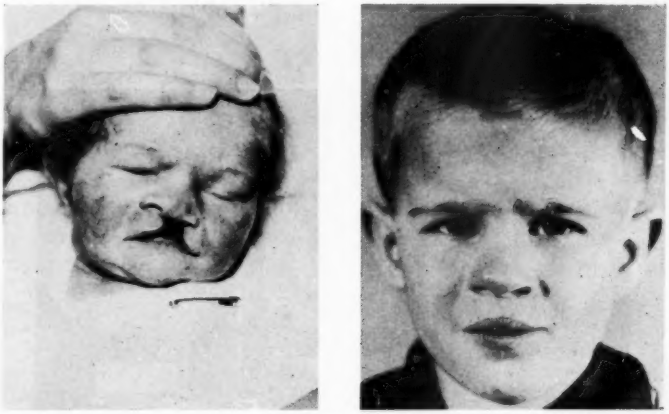

Fig. 80. Complete unilateral cleft of lip and palate, before and after surgery.

anatomic relation. The mucous membrane is realigned in the floor of the nose, and the skin from the external nares down to the mucocutaneous border is repositioned as a separate portion of the surgical approach. The muscles of the lip are detached from positions of anomalous insertion and aligned as a simple approximation in the line of the cleft. Best results are apparently obtained if the surgeon appreciates the embryonic derivation of the various elements in the normal lip and utilizes this knowledge in cleft lip surgery.

If properly executed, repair of the lip produces minimal scarring (Fig. 80). The new muscle forces established by repair induce striking and relatively rapid changes in the architecture of the alveolar ridge and palate. These changes include an approximation of segments toward

Fig. 81.

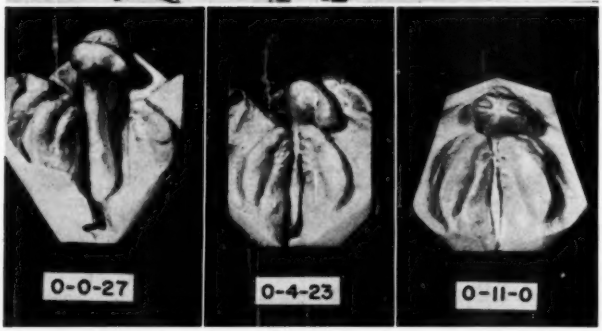

Fig. 82.

Fig. 81. Two-stage procedure for repair of a bilateral cleft lip. *A* and *D* reveal the deformity prior to lip repair. *B* indicates the conversion into a unilateral cleft, and *C* and *E* the result following complete repair.

Fig. 82. Changes in the palate following the two-stage repair of bilateral cleft lip. The initial cast was obtained at 27 days of age prior to lip repair. The second cast followed repair on the left side, and the final cast was obtained after complete closure. Note the molding actions of the lip and the narrowing of the cleft in the hard and soft palates.

the midline and a narrowing of the lateral dimensions of the cleft in the palate (Fig. 79).

Surgery of the bilateral lip deserves additional comment. It is frequently a difficult technical procedure and is often performed in two stages. The two-stage procedure is carried out by repairing one side and thus turning the deformity into a unilateral cleft. After an interval of

at least three weeks, in which repair is stabilized and circulation fully established on the operated side, the second side is repaired (Figs. 81, 82). Advocates of the two-stage operation point out that it is in keeping with principles of plastic surgery by conserving the blood supply of the prolabium.

A complicating feature of the bilateral cleft lip is the characteristic projection of the prolabial process and premaxilla beyond the tip of the nose and a markedly foreshortened columella. Not only do these features render repair of the lip difficult, but also even after successful repair the columella remains short, the nose flat and the upper lip protrusive.

Since the cosmetic results immediately following repair of the bilateral lip may be less than desired, it is significant to note that serial

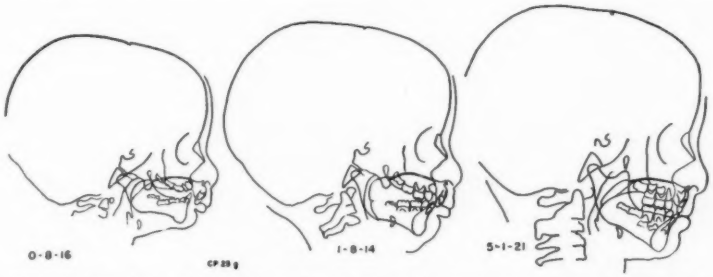

Fig. 83. Tracings of serial lateral cephalometric roentgenograms of a complete bilateral cleft of the lip and palate. At 8 months and 16 days of age, after complete repair of the lip, the premaxilla continued to protrude. Slight reduction of the protrusion was observed one year later. By 5 years of age, and without any form of intervention on the part of the surgeon or orthodontist, a dramatic improvement in facial profile was recorded.

studies have indicated that the differential processes of facial growth tend to minimize the deformity and provide a more desired end result.[6] In some children such spontaneous correction of the premaxillary protrusion may occur rather early and in others at a later age (Fig. 83). Of course there are rare exceptions in which there is no such improvement in the facial profile. In such instances section of a portion of the nasal septum may be required. In our opinion such procedures are to be used only as a last resort and are not to be regarded as an elective or alternative feature of primary repair of the bilateral lip. Further, the decision for section of a portion of the vomer should be based upon documented serial observation, such as is obtained by cephalometric roentgenography, over a period of at least five years.

An explanation for the progressive and continued improvement in the facial profile and in the shape of the nose and lip can be found in an analysis of the aberrant muscle forces that characterize the bilat-

eral cleft lip. The premaxilla and maxillary processes grow in an environment that is strongly influenced by the molding forces emanating from the contractions of the muscles of facial expression. In the bilateral cleft lip there are no such restrictive forces across the midline, and this fact accounts for the physiognomy of this deformity. Surgical repair of the lip alters the direction of these muscle forces by restoring continuity and symmetry across the upper lip and by the containment of the bony elements of the upper jaw within a functional muscle environment. This is as far as surgery can and should proceed. After having established a new environment in which growth can forge ahead, further resolution depends on the individual pattern of facial growth to minimize the deformity. In this instance, as in others, surgical judgment depends in large measure upon a knowledge of normal and abnormal patterns of facial growth.

Overmanipulation or improper placement of tissue in the primary repair of the lip may produce an immediate result that is more satisfactory from a cosmetic viewpoint. But because of the diminution of growth in these structures by such surgery, the ultimate result is the production of a deformed nose, face and jaw.[1]

CLEFT PALATE

Palatal Surgery

In the past the rationale for repair of clefts of the hard and soft palates was based on the assumption that the problem was only that of closing a defect or hole in the roof of the mouth. Accordingly, ingenious and difficult operations were devised in which soft tissue covering the palatal plates was displaced medially or else everted as a flap to cover the open defect. Other methods used flaps of bone and soft tissue from the palatal plates. Compression methods involving fracture and wiring of bone were attempted as a means of approximating the bones of the upper jaw and reducing the lateral dimensions of the cleft. Plastic procedures utilizing tissues taken from the cheeks, the pharynx and from extraoral sites were carried out as a means of repairing the cleft.

Two things went wrong in so many cases in the past as to warrant re-examination of surgical methods and philosophies. First, many of the patients had deformed faces as they grew older. The characteristic dished-in face became the stereotyped result of a generation of surgical experimentation. Secondly, and most important, normal speech was not with any consistency attained.

The newer knowledge pertaining to the growth of the face in man derived from the introduction of cephalometric roentgenography by

Broadbent[2] prompted similar analyses of the cleft palate problem. Cross-sectional studies in which operated as well as unoperated cleft palate cases were compared to normal standards revealed that the unoperated cleft compared favorably in terms of facial development with the normal. On the other hand, the operated cases showed varying deviations from the normal in the development of the face. The greater the number of operations performed and the earlier the age at which the palate was repaired, the greater was the deviation from the normal.[3] The results of recent studies demonstrate findings indicating that atraumatic palatal surgery, if judiciously planned and well executed, need not interfere with the growth processes of the face.[7, 8]

More recently longitudinal growth studies beginning with infants have indicated the wide range of variation in the severity of the deformity.[4] These investigations have emphasized the need for developing diagnostic criteria upon which the success of palatal surgery may be prognosticated. The following anatomical and physiological factors have been suggested as measures for estimating the suitability for surgery:[5] (1) width of the cleft, (2) adequacy of tissue adjacent to the cleft, (3) length of the soft palate in relation to the nasopharynx, (4) configuration of the nasopharynx, (5) the functional activity of the palatopharyngeal muscles, (6) changes in the foregoing criteria as a function of growth.

Width of the Cleft

Obviously, the wider the cleft in the palate, the more difficult its repair. However, the width of the cleft is not always a constant dimension. In certain clefts the width may not be altered as the infant grows older. In a few it may even grow wider, but in most it narrows to a greater or less extent. A cleft palate may narrow either through repair of the lip, changes in the posture of the tongue, growth of adjacent parts or by a combination of two or more of these mechanisms.

Repair of a unilateral cleft of the lip and palate may produce such extreme narrowing of the cleft that palatal surgery is greatly facilitated at an early age (Fig. 84). In other instances the narrowing may be less extreme, but may continue to narrow in time so that more optimal conditions for palatal surgery obtain at some later date (Fig. 85). Although approximation of segments and narrowing of the cleft in the palate inevitably follow repair of the lip, the changes are not always sufficient to produce favorable circumstances for palatal repair.

The tongue has been implicated as a factor in producing excessively wide clefts, particularly when mandibular micrognathia is a concurrent finding and the tongue is postured within the nose and nasopharynx. As the micrognathia is reduced by the further growth of the mandible

Fig. 84.

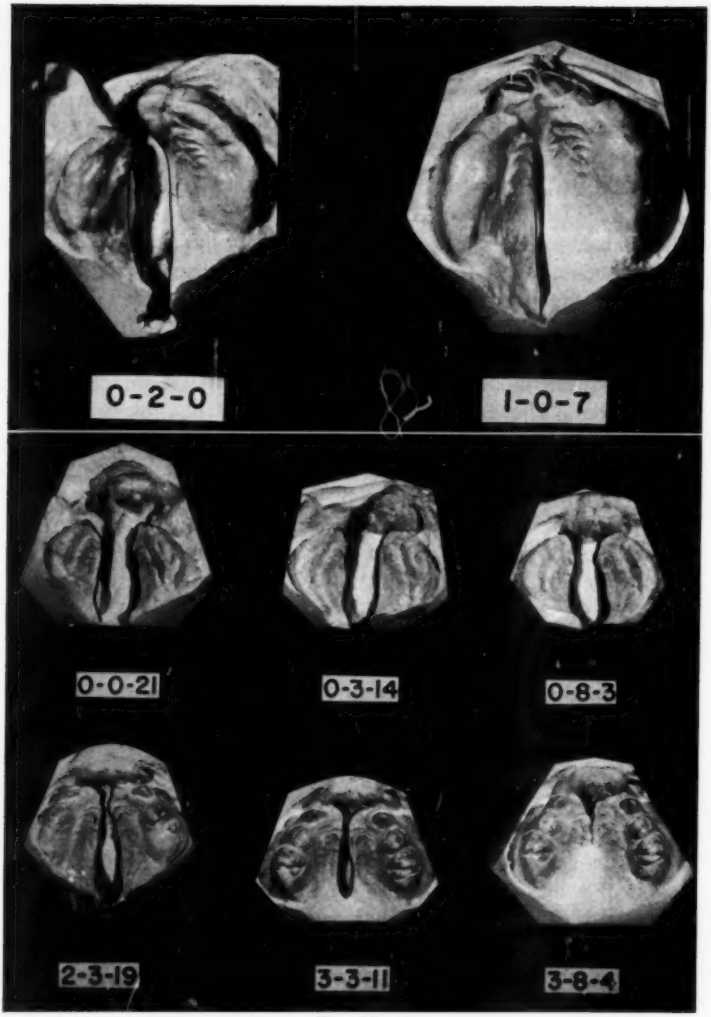

Fig. 85.

Fig. 84. Changes incident to lip repair of a complete unilateral cleft of the lip and palate. Note the extremely narrow cleft and the relative abundance of palatal tissue.

Fig. 85. Complete bilateral cleft of the lip and palate. The serial casts range in age from 21 days to 3 years, 8 months and 4 days. The first 3 casts reveal the changes following a two-stage procedure for repair of the lip. The fourth cast in the series shows continued narrowing in the lateral dimensions of the cleft. The results of repair of the soft palate followed by repair of the hard palate are shown in the last 2 casts.

and the tongue assumes a lower posture within the oral cavity, there is the concurrent finding of a narrowed palatal cleft. These observations strongly suggest a cause and effect relationship.

Growth of the palatal processes favors surgery first, by virtue of their relative increase in size in relation to the dimensions of the cleft. Thus more tissue is available that can be repositioned to cover the defect. Secondly, the enlargement of the palatal processes of the maxilla may contribute to an actual reduction in the lateral dimensions of the cleft, and by the increase in the depth of the palatal vault more optimal circumstances for palatal repair are available.

When is a cleft narrow enough to allow for surgery? When is a cleft so wide as to contraindicate surgery? Such judgments depend upon the experience, skills and attitude of the surgeon. However, certain generalizations with respect to such judgments seem warranted. The wider the cleft, the more extensive the operation required, and hence the greater the scar and potential restriction to growing structures. Finally, longitudinal observation is suggested as a method for determining the individual pattern of growth and change in the dimension of the palatal defect.

Adequacy of Tissue

Estimates as to the quantity and quality of the tissue adjoining the cleft are significant in judging the operability of the defect. As yet no acceptable quantitative measures have been presented to estimate differences between the several kinds of clefts and within the same kind of defect. Nevertheless clinical experience clearly demonstrates great variance in the quantity and quality of the tissues which are adjacent to the defect and which serve to provide tissue for its repair.

Length of the Soft Palate

In seeking to establish functional occlusion of the nasopharynx during speech and deglutition as a result of palatal repair, the length of this organ in relation to the nasopharynx is crucial. To obtain a preoperative estimate of its length, cephalometric roentgenography and laminagraphy offer a most useful diagnostic aid. As standards for the normal soft palate become available, measurements of the cleft palate group will become more meaningful and the prognostication of surgical success will be assured.

Configuration of the Nasopharynx

The dimensions of the nasopharynx, both in anteroposterior depth and lateral width, are important variables influencing the possible

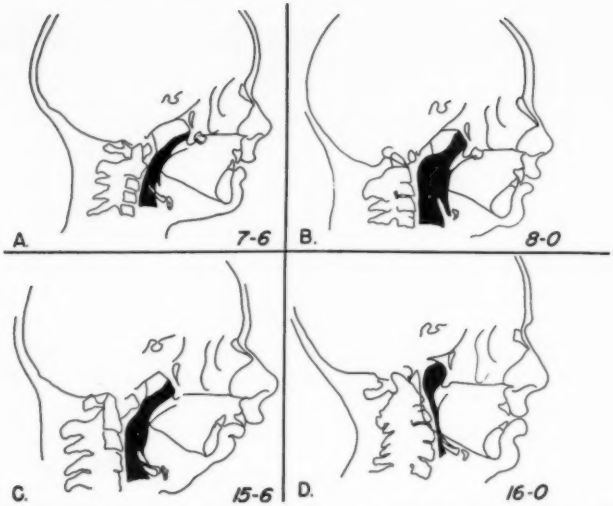

Fig. 86. Varying dimensions of the nasopharynx shown in tracings of lateral cephalometric roentgenograms. *A*, Normal child, age 7 years and 6 months, with intact adenoid. *B*, Occipitalization of the atlas increases the anteroposterior diameter of the nasopharynx. *C*, Atlanto-axial dislocation. *D*, Cervical lordosis subsequent to poliomyelitis.

success or failure of surgical repair. A method of estimating the width of the nasopharynx in the living from cephalometric laminagrams has been published.[9] The anteroposterior dimension can be evaluated from a lateral cephalometric x-ray film (Fig. 86).

Functional Activity of the Pharyngeal Muscles

The compensatory activity of the muscles of the lateral and posterior pharyngeal walls, apart from the length and mobility of the soft palate, are important factors in determining the ultimate capacity of the patient to effect palatopharyngeal closure. This can be done by direct inspection of the pharynx in the unanesthetized subject. The analysis of these variables as a preoperative routine and as a guide to the choice of surgical technique is recommended.

Changes with Growth

Growth, by virtue of the changes it may induce in the relative size of parts adjacent to the defect or by neuromuscular adaptations, may provide altered circumstances more or less favorable for palatal repair

at some later age. Hence periodic observation, preferably recorded by means of x-ray films or casts, affords some measure of the patient's increased or decreased capacity for palatopharyngeal valving. This potential for change with time, as a function of growth, should be a guiding factor in the selection of appropriate surgery of the palate.

Surgical Procedure

The surgical problem of repairing a congenital cleft of the palate is well known and is fraught with such controversies that any attempt at a comprehensive review would only lead to further confusion. A more logical approach would be to rely on the information emerging from growth studies of these children as a means of separating fact from empiricism and rigid dogma. On the basis of documental evidence available so far, the following general principles emerge as useful guides:

1. The majority of clefts of the palate are susceptible to successful repair by surgery. However, where there is an actual deficiency of tissue or where the tissue is so placed that it cannot be utilized in proper reconstruction, it is best to rely on a prosthesis as a means of temporary or permanent habilitation.

2. In some cases closure of the entire palatal cleft is feasible as a one-stage operation. Other patients are best managed by repairing the palatal cleft in two or more stages. Closing the posterior aspect of the cleft as a primary procedure has been advocated on the grounds that it reconstitutes normal muscular balances in the palatopharyngeal complex as soon as possible.[8] Furthermore this procedure prevents the posturing of the tongue within the nasal cavity and by such changes influences the development of a more normal bony architectural pattern within the maxillary framework. After a lapse of time the remaining portion of the cleft may be repaired, or the opening may be allowed to persist, in which event the residual cleft may be obturated by means of a prosthesis (Fig. 87), allowing the soft palate to function freely.

3. In order to compensate for deficiencies in length of the soft palate, push-back procedures for extending its length within the nasopharynx have been devised. Flaps based on the posterior wall of the pharynx have been elevated and attached to the soft palate to serve as tissue obturators of the nasopharyngeal aperture (Fig. 88). The push-back procedure and the palatopharyngeal flap have been used in combination for the more difficult cases. Although considerable enthusiasm has been generated for such surgical procedures, there is no evidence that they offer a surgical panacea for all patients. Further evidence, including carefully documented preoperative as well as postoperative analyses and

long term follow-up, will be required before the full value of such surgery can be appraised.

4. The end results of palatal surgery, in terms of its contribution to normal speech, facial growth and dental occlusion, cannot be fully evaluated until the child has matured into a young adult. This is due to the fact that the child is a moving target, and while we aim for

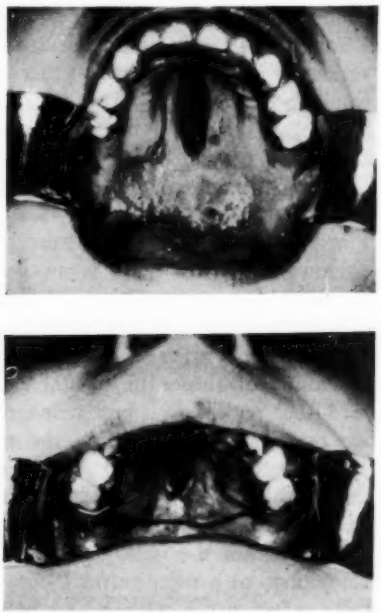

Fig. 87. Use of a prosthesis as an interim aid to speech. A, After repair of the soft palate. B, Clear plastic appliance to obturate palatal opening. Subsequently the remaining cleft was successfully repaired.

success at one point in his growth, our sights must also be adjusted to his future maturation. For these reasons a knowledge of the processes of facial growth and the documentation of individual growth patterns become essential features of surgical diagnosis, treatment planning and follow-up care.

5. There is no single rule for the timing of palatal surgery that is applicable to all patients. Ideally, it would be best to effect a complete repair at an early age so that normal muscular forces be established and the development of speech not be penalized by the presence of an

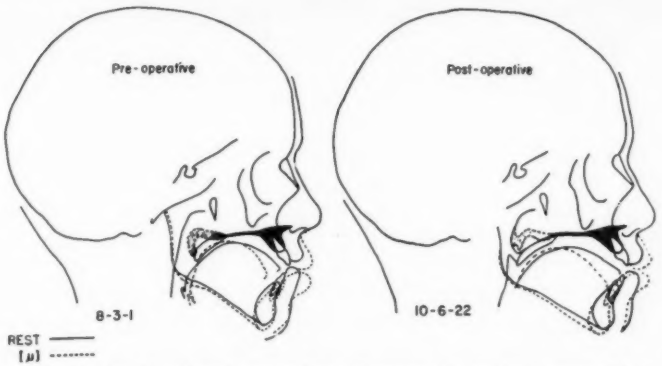

Fig. 88. Results of palatopharyngoplasty. Preoperative tracings supported a diagnosis of congenital palatopharyngeal incompetence. Although the soft palate elevated during the sustained phonation of the vowel *u*, it did not effect complete closure. The pharyngeal flap contributed toward nasopharyngeal valving and did not impede the previously recorded range of velar elevation.

open cleft. Unfortunately, ideal circumstances favoring early repair exist in only a small percentage of cases.

In our experience it has been possible to repair the soft palate in a number of patients by the age of one year. If such is carried out without excessive undermining of periosteum, destruction of blood supply or fracture of bone, there is no evidence of disturbed growth.[8] However, *such early procedures should not be performed in all cases.* Prescription of surgical treatment, in kind and in time, rests on an analysis of the severity of the deformity.

Orthodontics

Orthodontic follow-up and treatment is an essential service for many patients. In some it is a necessary preliminary to further cosmetic or prosthetic care. The age at which orthodontic treatment begins varies with the child and the problem.

Repair of the lip, in cases of cleft lip and palate, often induces overriding of palatal segments with moderate to severe asymmetry in the maxillary arch and palatal vault. To restore symmetry of contour in the alveolar ridge and hard palate, treatment can be initiated as early as three years of age. Such orthodontic manipulation is designed to reposition the palatal segments and provide optimal conditions for the growth of the bones and the eruption of the teeth. Improved dental occlusion and the facilitation of articulate speech are important objectives of such therapy. For the surgeon oral-orthopedic treatment is important, for it provides a symmetrical facial skeleton as a foundation

for such additional cosmetic surgery of the lip and nose as may be required.

Speech Training

Almost all persons suffering from a cleft palate defect can benefit in varying degrees from speech training, regardless of the success of surgical closure. Speech training is usually started at four years of age so that the patient will have the advantage of some instruction before entering school. Treatment integrated with the school curriculum or special intensive summer courses or a combination of the two is utilized in order to obtain the maximum results. The mentality of the child, the degree of the defect, intelligence of the parents and the success of the surgeon, orthodontist and prosthodontist are all factors in determining the results that will be obtained by the speech therapist.

CONCLUSION

Evidence is at hand that surgery can and does inhibit normal growth. This interference is directly proportional to the amount of injury to growing tissues and to diminution of blood supply to the parts concerned. Other evidence is available to show that the faces of children with clefts, unless permanently damaged, grow at normal rates. These two findings prompted a re-examination of concepts and procedures in surgery for cleft lip and palate. Together, they indicated that surgery poorly executed or poorly timed can do more damage than good in the long run.

Recent studies revealed that careful selection of patients according to certain diagnostic criteria and followed by conservative and properly timed surgical procedures assures greater measure of success in terms of good speech and proper facial growth. Prosthetic speech appliances offer an important alternative to surgery for clefts of the palate that are deemed inoperable.

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St. Francis Hospital
Evanston, Illinois

PSYCHOLOGICAL AND PSYCHOPHYSIOLOGICAL ASPECTS OF ORAL ACTIVITIES IN CHILDHOOD

DANE G. PRUGH, M.D.

THE BIOLOGICAL FUNCTIONS OF THE MOUTH

From a phylogenetic viewpoint the mouth is one of the oldest and most basic organs among the human being's biological pieces of equipment. The function of ingestion of nutritive materials is carried out by an organ resembling a mouth in organisms far below the vertebrate level in the evolutionary scale. The presence of a separately movable lower jaw, jointed to the skull, is a characteristic feature of the "body plan" of the vertebrate classes in the phylum Chordata, with the single exception of the primitive "jawless" fishes.[26] In mammals and other classes of walking vertebrates the mouth, through its connection with the pharynx, serves as the entrance to a "common corridor" for crossing pathways for air and food.[2] For man, the associated laryngeal structures enable the mouth to participate mechanically in speech as well, thus facilitating intellectual and social development.

In the human infant certain aspects of the biologic significance of the mouth and associated structures are clear. This organ serves immediately as the avenue for ingestion of food and, within several months after birth, as the site of beginning digestion, through the action of salivary enzymes. The strength and efficacy of the infant's sucking, swallowing and rooting reflexes, all of which should be present and intact at birth, are important influences upon his chances for survival. To a certain extent, particularly during crying or any period of

From the Departments of Psychiatry and Pediatrics, University of Rochester School of Medicine and Dentistry, and Strong Memorial Hospital, Rochester, New York.

temporary obstruction of the nasopharynx, the oropharynx participates also in the involuntary act of respiration. The infant's needs for the satisfaction of hunger are signalled by the reflex action of crying, which thus subserves a kind of primitive communication between infant and mother. Later, as the deciduous teeth appear, mastication of food is possible. Still later the use of the mouth and associated structures makes possible the articulation of words in the course of the development of speech and the complex act of symbolic communication.

A careful scrutiny of the neonate and the feeding situation brings to light other aspects of the biologic meaning of oral function. These include certain facets of behavior which are included in the broader definition of human biology emphasized by Romano.[24] The function of the infant's cry as a kind of signal of distress has been mentioned. In addition, it is apparent that the infant's first social relationships center around oral activities. Long before the infant develops any conscious awareness of the mother as a person or of himself as an individual, a kind of social and emotional "transaction" or reciprocal interaction[9] appears to take place between mother and infant. Through his indication of his hunger by crying the infant stimulates a response on the part of the mother. Through her proferred feeding by breast or bottle, with associated fondling and emotional warmth, the mother brings about a relief of the tension of hunger and an apparently pleasurable sensation on the part of the infant. Thus the feeding situation ideally comes to be associated with a feeling of satisfaction and security for the developing infant, as the result of the nutritional and emotional supplies given by the mother.

It has often been overlooked that the nature of the response of the infant to the feeding approach is of vital emotional significance to the mother. Most observers concede the immediate and long-range importance of the emotional "supplies" given to the infant by the mother during the act of feeding. It is easy to forget, however, that the mother herself requires certain evidence of positive response from the infant, in order to maintain her confidence and security as a mother. Thus the "feedback" of indications of pleasure and satisfaction from the infant to the mother renders a symbiotic quality, centered around the feeding situation, to the relationship between the two. The infant is of course completely dependent upon the mother or mother-substitute for basic satisfaction of his needs and even for survival. Nevertheless, in an emotional sense, each needs the other, and an emotional transaction does appear to take place.

In a very real sense this symbiotic relationship and the underlying emotional transaction between mother and infant can be said to form a facet of their original biologic relationship, begun in utero and continued, on a differing basis, after delivery. If the infant fails to receive

at least a minimum of emotional warmth from the person feeding him, he may well refuse feedings, thus endangering his nutritional state. If the mother fails to receive some emotional feedback (from an infant with a weak sucking reflex or a largely apathetic type of response), her capacity to provide such continuing emotional supplies may be influenced adversely, affecting her feeding approach, the infant's response, and again the infant's nutritional state.

At a later point in development the mouth assumes other functions. During the first few weeks of life it becomes apparent that the infant requires a certain amount of sensory pleasure from the act of sucking, over and beyond the basic relief of the tension of hunger produced by the feeding itself. This "sucking need" first shows itself in the infant's occasional, rather desultory mouthing of the nipple of breast or bottle, during moments when hungry sucking ceases. Later, after development of hand-to-mouth coordination during the second or third month, most infants exhibit finger or thumb sucking at times when hunger is not present. Thus the function of the mouth as a source of pleasurable sensory stimulation appears to "precipitate out" from the original sucking behavior which specifically subserves nutritional intake. The capacity to distinguish taste sensations, assisted by olfactory equipment, appears early in primitive form, developing gradually, with associated emotional influences, into the sophisticated palate of the older child.

By the third month (or at times earlier) most infants have progressed in neuromuscular maturation to the point at which they are able to coordinate successfully tongue and hard palate movements in such a way as to carry a bolus of strained food to the pharynx and swallow it (retrusive reflex). Prior to this time infants tend reflexly to protrude food from the mouth with the tongue. By seven to nine months of age, licking or chewing movements are possible. If deciduous teeth have begun to appear, chopped foods may be handled successfully. Soon the infant enjoys chewing on a number of objects, many of which are decidedly non-nutritional in character. The nine months old infant who puts "everything" into his mouth seems to be obtaining pleasure from sucking or chewing on objects. At the same time it is apparent that the mouth is now used as a sensory organ of investigation. Through this approach the infant seems to obtain an impression of the size, shape, texture and consistency of objects, impressions which are not yet available to him through other avenues of tactile exploration.

At birth and for some time thereafter the infant possesses an exceedingly primitive and relatively undifferentiated set of emotional responses. Two basic patterns appear to be present. A pleasurable response, mentioned earlier as being of importance to the mother's emotional equilibrium, embraces principally a kind of blissful, dreamy

state which ordinarily precedes sleep. A negative or "unpleasurable" response ordinarily involves fretting or fussing, followed often by crying and, if satisfaction is not forthcoming, restlessness and random motor activity. (Urination, defecation and vomiting may easily ensue if the state of hunger or discomfort remains too long unrelieved.) Thus the lack of differentiation of emotional response results in ready, nonspecific and diffuse participation by oral structures, cardiorespiratory, gastrointestinal and genitourinary organ systems in the limited play of emotional expression available to the infant at this point in development.

From the second or third month on, however, most infants are able to smile in response to social contact, particularly from the mother. By the third or fourth month, cooing or beginning babbling is observed in association with pleasurable feeding or social situations. Initiation of social contact, involving spontaneous smiling on the part of the infant, ordinarily appears by at least the fourth or fifth month. Thus the infant gradually develops differentiated and appropriate types of emotional expression, of forcefully positive as well as of convincingly negative nature, involving oral (and facial) participation. This progresses, during the second half of the first year, to nonverbal (though nonetheless vocal) expressions of apparent feelings of joy, satisfaction, fear, anger, displeasure and vague anxiety. Such a developmental step proceeds of course in association with the increasing organization and integration of autonomic and cortical mechanisms which permit the infant greater capacities for postponement of immediate satisfaction of hunger and other needs, as well as enhanced ability to divert attention from inner bodily sensations to outer social and emotional interactions with other human beings.

During the latter part of the first year specific vocalizations appear which lay the foundation for the development of word sounds. Complicated emotional expression by means of verbal formulation of thought symbols thus gradually becomes available to the two year old child. By three years the child can ordinarily describe vividly his emotional state, including imaginative flights of fantasy which may be of joyous or frightening nature. The mouth thus takes its place at the highest level of the evolutionary scale, employing larynx, pharynx, tongue, cheeks, palate, jaw (and sinuses as resonating chambers) in the act of speech which, in the face of ongoing maturation of the cerebral cortex, makes possible communication and the learning of rational adaptive behavior. Crying of course remains for some time as the more specific and forceful expression of negative emotions, with the help of words (and, for the small child, occasional biting of other children or adults).

In summary then, the child's mouth and associated structures act as

organs of nutritional ingestion, taste, mastication and beginning digestion, sensory pleasure, tactile exploration, respiration (in a partial sense), social interaction, primitive and later symbolic communication, emotional expression and even as an organ of attack. The biological functions of the mouth thus clearly include somatic and psychological components.

In the older child and adult most of these functions are preserved and are employed at times concomitantly. The need for oral tactile exploration is of course replaced by more mature and effective techniques, available to the older child with more advanced tactile, motor and cortical development. The use of the mouth as an organ of attack is (hopefully) dropped out in the process of personality maturation in civilized societies, except under conditions of war or extreme duress. The mouth still retains in adult life its function of providing sensory pleasure, in situations ranging from the chewing of foods of appealing consistency and tastes, through the use of chewing gum, candy and smoking materials, to kissing and associated sexual activities.

PSYCHOLOGICAL AND PSYCHOPHYSIOLOGICAL COMPONENTS OF ORAL FUNCTIONS

Contemporary medicine recognizes the constant interrelationships among physiologic and psychologic processes within the psychobiologic unit—the human organism. All such processes contribute in complex fashion to the maintenance of physiologic and emotional homeostasis[7] and, in the child, to differing facets of growth and development, including personality maturation. Such a dynamic concept makes it artificial to regard the mouth and its associated structures as isolated phenomena. For the purposes of this symposium, emphasis will arbitrarily be laid upon the relationships between the functions of these structures in the infant and child and certain aspects of psychological development, with which they are intricately interwoven.

Psychological Concomitants of the Feeding Process

In the newborn infant the mouth performs the central (though not by any means the single) function of his existence—the act of sucking, which provides the only source of nutritient material. The feeding situation thus forms the cardinal point of contact between the infant and his environment. Initially the infant, operating principally on a subcortical level of behavior, responds to this contact through reflex responses to sensory stimuli. He responds reflexly by sucking to the stimulus of the nipple in his mouth or turns his head, in rooting reflex behavior, to the side of the cheek which is touched. Other stimuli from

the environment are of course perceived, including those arising from sound, from changes in temperature and position, and from motion, associated with the feeding process. Inherent differences are now known to exist among infants in their sensitivity to these various sensory modalities of experience. Thus the presence of tension or jerkiness in the mother's handling of the infant during feeding, related to her own fatigue or emotional tensions, may affect his total sensory response to the feeding situation, more markedly in some infants than in others.

By the age of two and a half to three months most infants begin to develop an ability to wait for variable periods of time before their hunger is satisfied. This change is partly predicated upon the ongoing organization and integration of autonomic and other bodily homeostatic regulatory functions. Blood sugar now fluctuates less wildly and other metabolic processes involved in the production of hunger operate less fluctuatingly than during the first one or two months. At the same time the infant develops a growing capacity for attention to other stimuli, including visual ones. These and other developmental steps appear to enable him to anticipate and later to await feeding—for example, by perceiving first the breast or bottle and later the mother's face.

In the face of these changes the infant gradually becomes able to postpone his immediate feeding satisfactions in favor of more advanced social and emotional interchange with the mother. From observations of this kind it seems clear that even the very young infant develops a kind of confidence in the fact that he will be fed and that feeding will be a pleasant experience. Furthermore, the infant appears to develop certain (principally conditioned) patterns of response to the feeding approach of the mother, initially with milk feedings and later with semisolid foods. The mother's confidence, warmth and reasonable freedom from emotional tension appear to enhance the quality of the "emotional climate" of the early feeding situation, with reciprocal influence by the emotional feedback from the infant.

Beginning with the third or fourth month, the infant becomes able to respond to the mother as a specific person and gradually is able to distinguish her from other persons. Thus the confidence in being fed becomes tied up with the pleasurable social response to the mother. Although the process is little understood, many investigators infer from observational data available that the infant develops his first conscious mental representations of the outside world around the mother in the feeding situation, probably in the form of a vague mental image which can be retained as a kind of visual memory for short periods of time. It appears at least that the infant's first specific perceptions of the mother as a loving person are intimately related to his mental impressions of her as a person who feeds him. (Other aspects of the mother's

participation in her infant's care contribute, in smaller measure, to these mental impressions.)

Beginning with the second half of the first year of life, the infant gradually gains a mental concept of himself as a person separate from the mother. By the tenth or eleventh month most infants are developmentally ready to feed themselves and to indicate definite food preferences, thus supporting the trend toward growing independence. The capacity of the mother to permit such independent steps is thus a positive force in personality development. Associated with such developmental steps is the normal appearance of so-called physiologic anorexia around the end of the first year of life, often continuing for several years. This drop in food intake appears to stem principally f om the slowing of the rate of growth after the first year, and persists ordinarily until more rapid growth sets in at the end of the preschool period.

From the foregoing material it is clear that the psychological concomitants of the feeding process and the quality of the early mother-infant emotional symbiosis exert an appreciable (though not unalterable) influence upon later emotional development. The early conditioned patterns of response to feeding are reinforced by the later conscious associations of the feeding situation with the mother and by the development of attitudes toward her. In this way the infant or young child receives an important positive impetus to his development of an out-going relationship with first the mother and later the father and other family members. Significant support for his development of independence stems also from the later handling of self-feeding.

The emotional climate of such early feeding experiences thus appears to be one of a number of important determinants of personality trends, particularly in regard to the child's approach to relationships with persons outside the family. Other factors include the biologic differences among infants in their sensitivity to varying stimuli, the nature of subsequent relationships within the family, and later events in the environment, interacting with the child's inherent capacities and predispositions. The multidetermination of behavior and development is a concept which is central to this discussion.

Appetite and the Significance of Food. Observations of later childhood development indicate that the importance of the emotional quality of the feeding process and the offering of food by the parent continues to remain in the foreground for a number of years at least. The receiving of food from a loved person comes to be regarded by the child as one important facet of the receiving of affection. Indeed, the eating of food at times of discouragement and apprehension continues to stand as a substitute for affection and support in many other emotionally healthy adolescents and adults. Nibbling at examination time and eating at times of despair are only two of the ways in which the

emotional significance of food appears to derive its quality and character from the prototype of the early feeding situation, beyond the nutritional value of the food itself. The importance of food in offering hospitality and the variation in concepts of the amount of food deemed proper for an infant or child, among persons of differing socio-cultural backgrounds, point up the additional influence of culturally derived attitudes toward food and eating.

It is apparent that appetite in the child is a most complex phenomenon. Physiologic processes such as the appearance of hunger pangs, with associated drop in blood sugar values, the current state of gastric function and the stage of development of the child are subtly intermingled with psychological attitudes toward food, the nature of associations with earlier feeding situations, the child's current emotional state, the current emotional state of the parent or other adult who offers food, the quality of the child's relationship with the parent, and other factors, including fatigue or illness.

Sucking Activities. The mouth and adjacent skin areas appear to be endowed with tactile sensitivity of higher degree and greater exquisiteness than any other bodily zone during the first year of life. Mention has been made of the very young infant's need for sensory pleasure in sucking, which becomes distinct from the act of sucking for nutritive ingestion. Recent studies have indicated that sucking of the thumb or fingers, which begins around the third month of life, continues in the majority of children until nearly three years of age. Sucking at this later stage ordinarily occurs during periods of anxiety, shyness, fear, fatigue, boredom, or at night preceding sleep. (Such night-time sucking is often associated with the fingering of a blanket or other soft surface or occasionally with twisting the hair.) A number of well adjusted children continue mild and occasional sucking well into the fourth or fifth year. It has frequently been noted that a child who has given up thumb sucking will suddenly exhibit a transient, regressive reappearance of such a need, in the face of later emotional stress or conflict. This development often occurs after illness and hospitalization—for example, in children of three, four or five years of age (usually accompanied by other signs of emotional regression or manifestations of anxiety). In older infants who have given up the bottle a transient, regressive return to such sucking satisfactions may be seen under similar circumstances involving separation from the mother.

For a number of years thumb sucking was strongly disapproved and vigorously combated by parents and professional people alike. The impression was held that thumb sucking frequently caused displacement of the upper incisors and premaxillary bone.[15] Certain studies[16, 19] have indicated more recently that, although temporary and reversible displacement of mild degree may occur in children with deciduous

teeth who suck their thumbs or fingers frequently and vigorously, lasting dental or premaxillary distortion ordinarily does not appear if sucking is discontinued prior to the eruption of the permanent teeth. (Other causes, including congenital or hereditary influences, have been felt to be more importantly involved in a number of cases of such displacement.) Since most children do give up sucking before this time and since sucking has been recognized as representing a specific emotional need in the young child, most professional persons now feel that no attempt to discourage such behavior need be made. Indeed, attempts at mechanical restraint or vigorous dissuasion frequently strengthen the habit and may endow it with a secondary meaning of passive resistance.

A similar distrust of pacifiers or "sugar nipples" existed for many years. It seems apparent today that the moderate use of a pacifier in the first several months of life produces no harmful effects upon later emotional development. Indeed, Levine[14] has reported its successful use in the treatment of colic in infants.

Considerable controversy in the past was carried on around the subject of weaning. It was felt that late weaning might predispose to greater dependence or to persistence of infantile habits. Abrupt or early weaning was regarded as causing greater thumb sucking or specific emotional trauma. At present no specific evidence exists to indicate that later weaning, within reasonable limits, causes serious emotional deviation. Conversely, no clear-cut evidence currently indicates that early or abrupt weaning always produces strong sucking tendencies or serious emotional disturbance.[21] Very early or extremely abrupt weaning, in combination with other disturbing events, does at times seem to be importantly involved in such effects. The possibility of disturbance in feeding behavior also seems to be significantly greater under such circumstances.

Many infants will respond readily to weaning, carried out gradually over a period of several weeks, by the latter part of the first year, if the cup is offered between five and seven months. Some infants cling to the bottle, however, until the middle or latter part of the second year or even later, without signs of deviation in emotional development. The relationship with the mother appears to be the most important single factor in the determination of the response to weaning and may also determine the way in which the weaning is carried out.

Mastication. There is some indication that the pleasure associated with the chewing and biting of food, after the eruption of the deciduous teeth, is important for emotional development during the early phases of infancy. As the infant develops, chewing, biting and swallowing later assume other psychological meanings. The primitive use of biting as a means of defense or attack has been mentioned. At two or

two and a half years many young children exhibit normally mingled or ambivalent feelings of affection and resentment toward a parent by first hugging, then playfully biting the loved person. Thus biting in particular comes to be associated with the presence of aggressive impulses or hostile thoughts, to the point at which many children of this age may fear temporarily being bitten by harmless objects, as if in punishment for such thoughts or feelings.

At the age of three or four years, with the enhanced development of speech, children normally exhibit variously illogical and exceedingly primitive thoughts or fantasies about "biting" or "eating up." Thus the three and a half year old child talks freely about the big bad wolf who "eats up bad little boys," a theme common in fairy tales and comic books. When he feels affectionate, he may say to his mother, "I love you so much I could eat you up" (a type of expression not far removed from adult ones). At times of anger he may threaten the parent or another child with being eaten up or may pretend to be a bear and declare his intention of eating up the parent. At times, overexcited or anxious children may become frightened of playful fantasies of this kind, which become almost too real. In addition, children of this age, already aware of sex differences, may include among their hypotheses regarding the origin of babies the (ordinarily transient) belief that the mother swallows the "seed." In most instances the child's capacity to distinguish fantasy from reality gradually increases by the age of four or five years to the point of predominantly rational control.

When the deciduous teeth begin to fall out, the six or seven year old child normally exhibits a great deal of curiosity mingled with some fear over this process. Pride in "growing up" eventually predominates, but some children exhibit apprehensions about this process in keeping with their tendencies still to fear bodily alteration or mutilation as a kind of punishment at this stage. (Such tendencies toward misinterpretation are also involved in the otherwise obscure fears of four or five year olds of having their legs cut off or "something else" taken out when a tonsillectomy or other operation must be performed.)

From the foregoing material it may be seen that the psychologic concomitants of oral activities are extremely pervasive in emotional development during early childhood. Space does not permit a full discussion of the psychological significance of certain other oral functions, including that related to the act of respiration. Brief mention should be made of one specific item. The function of speech is exceedingly important and complex. The normal and almost universal tendency toward the occasional repetition of words and hesitation in speech by the two and a half or three year old child, in situations ordinarily involving complicated concepts which are difficult for him to express, should be kept in mind as a developmental phenomenon

ordinarily transient. It remains to indicate the mental association of certain words at least with aggressive impulses or hostile thoughts. Such an association commonly takes place during the period from three through five years, when words are regarded by the child as concrete and almost magic symbols which, if spoken, might cause the associated thought or fantasy to "come true." Ordinarily, those primitive concepts are brought under control with the advance into the school-age period and cause no difficulty.

Psychophysiologic Aspects of Oral Functions

With the growth of knowledge regarding the interrelationships between somatic and psychological processes has come a recent interest in psychophysiologic considerations involving the mouth and related structures. Much of the work which has been done in this area is of a decidedly preliminary character, and a great deal of investigation remains to be carried out. An attempt will be made, however, to summarize briefly certain pertinent data.

Among the functions of the mouth which have been investigated is the production of saliva. Studies have indicated that variations in amount, viscosity and pH of salivary fluid may occur in relation to variations in emotional state.[5] Evidence also exists that the blood supply to the gums through the capillaries varies, within slight limits, with emotional fluctuations. Psychological components are present in the neuromuscular activities of chewing, swallowing and other voluntary movements of the mouth and jaws, particularly in regard to the coordination of muscles involved in these activities. For example, the set of the jaws and the degree of muscular tension involved may determine, in some measure, the type of dental occlusion, along with other factors. Alterations in smooth muscle function, involving the lower two thirds of the esophagus, may affect the efficacy of the involuntary components of the act of swallowing, in accordance with emotional influences. Indirectly, of course, the reflex activity of the cardiac sphincter of the stomach is related coordinatively to swallowing movements of the mouth and esophagus. It is also well known that other reflex properties, such as the sensitivity of the gag reflex, may be influenced by psychological states. The efficiency of the laryngeal apparatus and, to a certain extent, the upper respiratory tract depends upon the intactness of involuntary neuromuscular coordination and the degree of tension present in muscular structures. Through the gastrocolic reflex and other mechanisms the ingestion of food may affect indirectly the activity of the lower intestinal tract. The influence of the emotions upon gastric secretion and the state of the gastric mucosa is well known.

This cursory review of the psychophysiological aspects of the function

of the mouth and associated structures illustrates, among other things, the importance of continuing investigations in the psychophysiology of bodily functions. A new physiology is being written, even on the basis of the limited knowledge at hand. Our limited understanding of pathological disturbances in function of psychophysiologic nature, however, still exceeds our even more limited grasp of the complex forces involved in the maintenance of normal functions. As yet the neurophysiologic pathways by which such influences are coordinated remain dimly understood. Cortical and autonomic interconnections have been implicated, among other mechanisms, in the transmission of emotional influences to various organs. However, *variations in vascular flow, salivary secretion, local reflex activities, the action of smooth muscle components of the upper gastrointestinal tract and voluntary and involuntary neuromuscular coordination do not take place in a vacuum.* Other changes in related organ systems throughout the body participate in such psychophysiologic fluctuations in the states of health and disease.[7]

PSYCHOPATHOLOGICAL AND PSYCHOPHYSIOLOGICAL DISORDERS IN ORAL FUNCTION

In the light of the foregoing discussion it is easily seen that a number of pathological forces may impinge upon psychological development as it relates to oral activities and upon the healthy psychophysiologic functioning of the mouth and its associated structures. The points at which such influences may come to bear will be briefly summarized and the types of deviations in function and development broadly indicated. Space does not permit detailed discussion of relevant psychodynamic considerations, which may be found elsewhere.

Disturbances in Feeding Behavior

Basic alterations in the feeding activity of the very young infant may occur within the framework of the emotional symbiosis and reciprocal interaction between mother and infant already described. Tension or emotional disturbance, illness or fatigue on the part of the mother, as well as adverse circumstances during early milk feeding, may influence the feeding behavior of the infant, which in turn may affect the mother's further functioning in this process. Conversely, illness producing anorexia in the infant may threaten the emotional equilibrium of certain anxious mothers, leading to increased urging of feeding, particularly around the time of introduction of semisolid foods.

Too early introduction of semisolid foods, before the consolidation of the retrusor reflex, may confuse the infant—and the mother—rein-

forcing earlier disturbances in the feeding situation or producing new ones. The present trend toward the offering of semisolid foods during the first few weeks of infancy has little physiologic basis. Abundant studies indicate that endogenous supplies of iron in the infant do not require additional exogenous sources until at least after the third month of life. Other nutrient materials are well supplied up to that point by milk feedings and appropriate vitamins. Pancreatic and other enzymatic functions do not appear to be fully effective during the first month or two. An occasional, very active or advanced infant, however, may appear to be more comfortable with semisolid feedings prior to the third month.

Refusal of feedings is the commonest response of the infant to such transactional influences. In prolonged or intense disturbances of this kind a conditioned pattern of feeding refusal may be set up which may carry over to the infant's beginning conscious mental associations of the mother with the feeding process. Thus a pattern of struggle over feeding may become a pattern of struggle with the mother. Later, this may become generalized to include the father. In the face of more basically unhealthy parent-child relationships such conflict may spread over into later areas of development, reinforced by struggles over self-feeding and bowel control, for example, leading to broadly negativistic or rebellious behavior patterns.

In extreme instances of feeding refusal marked interference with the infant's nutritional state and bodily development may occur. Situations of this kind may result in very young infants from the lack of an adequate and consistent mother-figure and associated components of emotional warmth, as in the case of the death or prolonged absence of the mother, without a continuing mother-substitute. As Bakwin[3] and Spitz[27] have pointed out, institutionalization of young infants has been known to produce this picture of depression and feeding apathy, originally called marasmus, with of course other psychological and physiological reverberations as well.

The process of teething, though unrelated to convulsions, high fevers and other problems, may produce some mild malaise and anorexia. Some feeding problems have arisen at such times, particularly in families in which much apprehension over eating and resultant urging of food may be present.

Subsequent alterations in feeding behavior may be the outcome of difficulties in the acquisition of self-feeding tendencies. Certain parents encounter difficulty in permitting the infant reasonable independence toward the end of the first year and may feel a need to continue feeding the infant far beyond the point of possible self-participation. Demands by the infant for such continued feeding and other dependent satisfac-

tions may persist into later stages, or the infant may begin a struggle for control of this function.

Disturbances in feeding patterns during the second six months of infancy and early childhood, as the result of social, economic and emotional factors, may, as Pollock and Richmond have indicated,[22] result in inadequate intake of iron in solid foods and the picture of nutritional anemia. Help for parents in dealing with the feeding problem of course is equally important as iron therapy.

Disturbances in feeding behavior may derive from other and later sources than those seen during infancy. Regressive disturbances in feeding patterns, associated with illness or hospitalization, may appear in children throughout the preschool period. Such developments are ordinarily transient. At times, however, in the presence of intense anxiety or guilt on the part of the parents or within the framework of previous disturbances in the parent-child relationship, continuing feeding struggles of the type described may be precipitated by such intercurrent influences. Depression may represent another source of alteration in feeding patterns. Prolonged separation from the mother, without a central, warm and continuing mother-substitute relationship, may produce such depression in older as well as younger children. Refusal of food or pronounced anorexia may occur. In other instances children may eat increased amounts of food, using this source of pleasure as a substitute for the more basic emotional satisfactions involved in the healthy parent-child relationship.

In recent years, studies of older children with marked obesity[4] and adolescents with anorexia nervosa[30] have pointed up the important influences of early feeding experiences in these disorders. Most children with these pictures have a history of disturbance in early feeding patterns, combined with a more basic lack of emotional satisfactions in the parent-child relationship. At times, food has been offered or urged by the parents in place of more important emotional warmth which they are unable to give. As Hamburger has indicated,[10] obesity with emotional components results from disturbances in appetite of varying nature, produced by chronic anxiety, depression, addiction to food as a substitute gratification, and other factors. Other determinants are of course involved, including inherited potentialities, family eating patterns, lack of energy output in exercise and intercurrent illness or emotionally traumatic events. Such children with marked obesity of nonhormonal nature (and few seem to fall into the hormonal category) are often dependent and immature, resisting diets strongly because of the associated deprivation of unhealthy emotional gratifications derived from the eating of food. Mild pictures of obesity associated with prepubertal changes in boys are of course another matter.

Anorexia nervosa is a serious disorder of psychological development,

in some instances bordering upon psychotic degree, involving sweeping psychophysiologic disturbance related to the associated cachexia. In many such instances, girls, who show the disorder most frequently, have failed to accomplish solidly the normal control of primitive illogical fantasies at the preschool level. With the onset of puberty and adolescence they may show regressive fears of heterosexual independence which include unconscious confusions regarding the sexual act and impregnation, including fears of swallowing a "seed." Refusal of food has of course other components than the misinterpretations of this kind involving oral functions. Depression, suicidal tendencies, suspicion of some harmful influence from food and a regressive revival of old patterns of passive rebellion toward the parents by refusal of food also operate importantly in this multidetermined symptom of exceedingly serious import.

In some preschool children feeding problems center around the child's difficulties in control of his aggressive impulses. Certain children refuse for a time to chew solid food, demanding puréed or "baby" food, in connection with their difficulties in accepting healthy independence (which may have been difficult for their parents to permit). In other instances the association of biting, chewing and swallowing with hostile thoughts has become so intense that unconscious fears of biting and chewing food appear, involving for the child the danger of awareness during these acts of such associated thoughts or fantasies. Such children may hold food in their mouths for interminable lengths of time, unable to chew or swallow it. Ordinarily these problems are seen only in severely disturbed children, although a number of young children show transient deviations of this kind.

Disturbances in Sucking Patterns

Certain deviations in sucking behavior are observed in which preschool children show pronounced needs for thumb or finger sucking, at times to the exclusion of other activities and accompanied by social isolation or withdrawal. Occasional children continue vigorous thumb sucking, with or without associated social withdrawal, beyond the usual time of giving up this need for sensory pleasure. In such cases permanent displacement of the upper incisors and premaxillary bone can occur, though apparently this development is not uniform in all such instances.[16]

Serious intensification or prolonged continuation of sucking patterns ordinarily is associated with a more basic underlying emotional disturbance in the child and with dependent or immature patterns of behavior. Biologic differences in sucking needs, early struggles over feeding, abrupt weaning and other influences play contributory roles,

but the central pathology is often found in the parent-child relationship and its vicissitudes. In such cases an adaptive device for the satisfaction of an emotional need has been used too strenuously and often at the expense of other, more important, socially adaptive behavioral devices. Thus thumb sucking, the adaptive device, itself becomes a symptom, in a fashion similar to that seen in certain types of predominantly physical illness.

It is necessary, however, to look beyond the symptom itself to the total adjustment of the child, as with any symptomatic disturbance in behavior. Occasionally one finds a child of school age who shows relatively healthy emotional development, but still retains the relatively isolated and "encapsulated" symptom of thumb sucking. In most instances the thumb sucking is only one of a constellation of symptoms of failure in adaptation, including enhanced dependence, possible difficulty in school adjustment or performance, social isolation or other, more serious manifestations. Here one must recall the (unhealthy) adaptive significance of the symptom. Aggressive measures to remove thumb sucking without offering more basic solutions to the child's underlying conflicts may result at times in its replacement with another and occasionally more troubling symptom.

Although severe and prolonged thumb sucking is ordinarily associated with emotional disturbance, as pointed out earlier, *the emotionally secure young child often sucks his thumb or fingers.* Certain parents have emerged from their reading of pertinent literature with the mistaken impression that their child is exhibiting some disturbance in behavior or some unmet emotional need if he sucks his thumb at all. The importance of the total picture is self-evident.

Related psychologically to disturbances in sucking behavior are certain deviations in infants and young children which involve mouthing, at times chewing and occasionally swallowing of numerous objects, principally non-nutritional. Little research has been reported from a psychological approach to the symptom of pica, involving such behavior. Nevertheless, clinical impressions of a number of cases[18] have indicated that unsatisfied early emotional needs are at least an important component in some instances of this behavior, which of course may lead to various types of poisoning. Again multiple forces may be involved, including inherent differences in sucking needs, mental retardation and other factors.

Speech Disturbances

During the period of development of speech numerous psychological and social influences converge upon the infant. Most of these, as has been mentioned, are taken in stride by the infant with adequate endowment and a satisfying relationship with his parents. In some

cases, however, problems may arise. Moderately delayed speech in an otherwise healthy infant may make certain parents extremely anxious, especially if they have strong needs for verbal and intellectual performance. Unconscious pressure may be put upon the infant who, within normal limits, says only a word or two by the beginning of the second year. Tension may thus arise on the part of the infant which actually interferes with his performance by reducing the relaxed and natural coordinative efficiency of the muscles of the mouth, larynx and respiratory apparatus. Struggles over feeding, weaning or toilet training may enhance this difficulty.

A few infants may attempt to adapt to emotionally disturbing situations or events by negativistically refusing to talk, even in the face of pressure, holding back and demanding (and often receiving from troubled parents) objects or help requested strictly in nonverbal ways. Such behavior is often seen transiently in infants who experience the birth of a sibling around the end of the first year. More rarely, difficulties may continue, under pathological circumstances, as when a child is seriously disturbed by prolonged illness involving frequent hospitalizations and separations from the mother. Such problems of course complicate developmental evaluation of the infant's mental abilities.

In addition to delay in speech development, stammering is a not uncommon symptom of distressing import. A full discussion of stammering is not contemplated here. Its origin may frequently arise during the period of developmentally normal speech hesitation and repetition referred to earlier. Parental apprehension and conscious or unconscious pressure, intercurrent emotionally traumatic events and other influences may perpetuate this difficulty. Involved also in some cases is an unusually strong fear of the loss of control of hostile feelings, described earlier as being frequently but transiently connected with the expression of certain words and associated feelings. In such instances children seem adaptively to repress from awareness any such unacceptable feelings, a mechanism which appears to be reinforced unhealthily by an inhibition or blocking of the capacity to express particular words which might lead to the revelation of such feelings.

Early feeding struggles and later significant difficulty on the part of certain parents in accepting the child's healthy expression of his normally aggressive impulses are often found in these situations. Other factors, however, including familial tendencies and mild congenital brain damage, may also be importantly involved.

Other Psychopathological Disturbances in Oral Functions

Most workers consider stammering a psychopathological manifestation rather than a specifically psychophysiologic one. The distinction is

based upon the fact that no local changes in physiologic function are ordinarily noted. Thus stammering from primarily emotional influences is often considered a hysterical conversion symptom, involving a central disturbance in innervation in a fashion which is still poorly understood. The same appears to be true of diminution in the sensitivity of the gag reflex, seen at times in hysterical personalities, as well as in hysterical aphonia, globus hystericus, hysterical trismus and certain hysterical types of oral pain, all seen predominantly in older children and adolescents. Such central changes in innervation, reproducible under hypnosis, seem to represent unhealthy attempts to solve emotional conflicts through symbolic means. Thus an inability to speak or to swallow, for example, may occur in situations in which to do so would intensify the conflict for the child, as a result of unconscious temptation to say unacceptable words or to bite or swallow forbidden substances. Such psychoneurotic conversion disturbances are often included in the term "psychosomatic," since alteration in bodily function is involved, usually of the voluntary components of the skeletomuscular system. More specific psychophysiologic disturbances, ordinarily deriving from alterations in autonomic innervation and involving visceral organs and "vegetative" functions, will be discussed in the next section.

It remains to mention certain influences upon psychological development of congenital or acquired somatic abnormalities in the mouth and associated structures. These problems, sometimes called "somatopsychic" to distinguish them from "psychosomatic" disturbances (in which emotional factors play predominant roles), involve principally the secondary emotional reverberations of a primary somatic defect. The long-range effects upon the child's confidence, his concept of his "self" and his body image, as well as upon his social adjustment, of various dentofacial handicaps, such as cleft palate and hare lip, have been discussed thoroughly elsewhere.[6, 17] The cosmetic implications of such defects and the associated disturbances in speech production are only part of the picture, since the significance of the defect to the parents and their capacity to help the child deal with his feelings about himself are exceedingly important. The special meaning to the child of surgery on the mouth or related structures, together with other considerations regarding the possible effects of hospitalization and of separation from the mother upon the young child,[23] have also been investigated, particularly in relation to tonsillectomy and adenoidectomy.[13]

Psychophysiologic Disturbances

In this section will be considered briefly certain pathologic alterations in physiologic function of oral structures which stem from emotional conflict or tension. As in the earlier section on healthy psychophysio-

logic function of oral structures, it must be stated that knowledge is limited at the present time. Nevertheless, certain data are available and will be sketchily summarized.

From the foregoing material it is apparent that the state of health of the child's teeth, gums and buccal mucosa may be directly influenced by emotional and behavioral forces. The occasional effect of intensive thumb sucking has been mentioned. Biting the nails, which appears transiently in a large proportion of well adjusted school-age children, may reach such intensity that local ulceration of the gums or other detrimental influence on periodontal tissues may result. Biting pencils or other objects may also be a factor. In such severe instances unsatisfied emotional needs for oral sensory pleasure, often displaced from sucking tendencies, are frequently a factor, together with unhealthy aggressive impulses which are discharged through such biting activity rather than through more socially acceptable channels.

Grinding of the teeth (bruxism) is often seen temporarily in infants during the early part of the second year, occurring often at night. In some instances such activity is continued into later childhood at least, producing at times disturbances in occlusion and occasional alveolar destruction. Undischarged and overintense aggressive impulses again appear to contribute importantly to such pictures, as may be the case in instances of chronic clamping of the jaws. Lip-biting, cheek-biting and tongue-biting are well known results of emotional tension.

Other channels of influence upon dental health have been said to derive from excessive amount of certain foods, such as carbohydrates, in the diet of children with intense emotional needs for such intake. Here the specific degree of influence is less readily apparent, since the unclear etiology of dental caries seems at the very least to be varied and frequently multiple. The same is true of possible hormonal influences which have been indicted by certain authors. It is clear, however, that negativistic resistance to adequate brushing of the teeth in certain children may stem from earlier struggles over feeding and other aspects of development.

Mention was made earlier of psychophysiologic aspects of salivary function and of blood supply to the mouth. Variations in amount of salivary secretion may occur as the result of prolonged emotional tension; diminution of salivary flow, the so-called dry mouth, is often associated with loss of appetite. Certain investigators have also raised the possibility that alterations in salivary constituents, deriving from emotional influences, may operate as contributory factors to dental caries and as predisposing forces in the appearance and persistence of certain oral infections. Disturbances in nutrition of the gums, through vasospasm of the involved capillaries, appear to result at times from

emotional tension, thus operating as predisposing factors in certain cases of gingivitis and other forms of periodontal disease processes.[5] Much further research is necessary to validate these impressions.

In most of the conditions described, psychophysiologic disturbances, occasionally progressing to structural changes over long periods of time, are present elsewhere in related or other organ systems.

Full discussion of a number of other pertinent psychophysiologic disturbances is not possible in this review. In certain cases of recurrent or cyclic vomiting, vegetative disturbances in function of the lower esophagus and gastric cardiac sphincter seem to arise, in psychologically predisposed children, from the presence of intense, unresolved and usually unconscious emotional conflict. Such conflict often centers around the act of swallowing or "taking in" particular types of food with special emotional significance or under emotionally charged circumstances. (The latter situation is often found in mild cases of school phobia in rather dependent and anxious children, when the fear of separation from the home and the parents is not prominent, but is expressed psychophysiologically by vomiting in the morning before school begins.) Cases of cardiospasm have also been described in which emotional influences predominated.

In some cases of persistent rumination in infants the normal regurgitative tendencies may be enhanced by the infant's unhealthy sensory pleasure, related to sucking and obtained from the mouthing of the regurgitated material, occurring in the face of an unsatisfying emotional climate in the feeding situation. Other factors, such as chalasia of the cardiac sphincter, may of course play causative roles.

CONSIDERATIONS REGARDING MANAGEMENT OF THE PSYCHOLOGICAL ASPECTS OF ORAL FUNCTIONS

Detailed discussion of the clinical management of the aforementioned developmental processes and deviations is not possible in this paper. It is worth while, however, to put into perspective certain statements already made and to mention several important concepts regarding management. For fuller treatment of these topics, other sources may be consulted.[8, 25]

For the pediatrician or general practitioner, work toward the prevention of emotional disturbances represents the most fruitful and effective utilization of his time. Mention has been made of more severe disturbances in adaptation which involve the oral functions. In many such instances intensive psychotherapy for child and parents will be necessary in order to resolve the pathological forces within the family and to permit the child to work out healthier solutions for the con-

flicts which produce tensions and symptoms. Milder problems may respond more readily to the investment of time and patient work on the part of the practitioner. Direct discussion of moderate thumb sucking tendencies in a school-age child who shows otherwise relatively healthy adjustment, for example, may produce positive effects by helping child and family to look at the problem more objectively and with less tension and concern. If the wish to give up thumb sucking is present, such an approach may help the child to control this behavior in situations in which orthodontic procedures are necessary.

In more serious personality disturbances the pediatrician or family physician may find that the family cannot accept referral or that no psychiatric facilities are immediately available. A supportive approach by the physician, involving frequent contacts and some discussion of superficial and presenting problems with parents and child, in an understanding and noncritical way, may carry such a family along adequately. (Such an approach necessarily does not involve the "operative" uncovering, by insight-producing techniques, of deeper anxieties and conflicts which the persons concerned are not yet ready to face.) Ultimate referral for psychotherapy may thus become possible or available.

If referral is contemplated, the role of the physician in helping parents to understand the emotional nature of the problem and in minimizing guilt and uncertainty can be a most formative one. The previous discussion regarding the principal sources of severe problems within the family relationships should not obscure the fact that such problems are not consciously produced. Even a seriously confused or strongly punitive parent ordinarily is usually consciously trying, as effectively as he can, to resolve a problem in his child, even though unconsciously he may be involved, among other forces, in its perpetuation. Unfortunately, some of the literature for parents and professional persons in recent years, while attempting constructively to clarify the sources of emotional disturbance, has left the impression that children's problems are the "fault" of the parents. All physicians need to employ profound humility and understanding regarding such problems. The practicing physician can do a great deal to dispel parents' misapprehensions and fears of blame and to aid them in mobilizing their own constructive forces in order to seek help for serious disturbances.

In regard to the preventive aspects of the role of the pediatrician or family physician, much has already been written.[11, 26] Some of this material has dealt with the importance of gentle examination of the mouth of the young child, the need for a positive relationship with a child before painful dental or other procedures are carried out, and the necessity for psychological preparation of the child for operative proce-

dures. The broader aspects of a psychological approach to prevention of emotional disturbance, related to oral or other aspects of development, have also been fully discussed. Through the support and wise guidance of the physician, many parents may find easier roads to rearing their children in the direction of healthy emotional patterns of adjustment. The principle of anticipatory guidance remains the central one in well-infant and well-child work. By giving the mother (and father) some knowledge of new developmental stages slightly in advance of their appearance, the physician can help them to deal with the newly emerging behavior, oral or otherwise, in reasonably ready and comfortable fashion. (Too much knowledge and self-consciousness about the parental role of course may exert a negative rather than a positive influence.) At the same time the physician can provide an atmosphere wherein the parent feels free to verbalize any uncertainties or confusions without fear of criticism. Families in urban society today are more mobile and have fewer consistent contacts with previous generations from which to draw models for their own child-rearing practices. The physician thus frequently must fulfill his important role as family adviser in an even more significant fashion.

Final mention should be made of certain aspects of feeding practice which have been widely discussed. These relate principally to the concept of more flexible[1] or "self-demand" feeding. Such an approach, like breast-feeding,[20] is becoming recognized again, after years of neglect, as having certain advantages for the infant. The more flexible approach does permit the gearing of feeding to the infant's initially fluctuating physiologic rhythms. As with breast feeding, many mothers enjoy the more natural response to the infant's needs, particularly during the first two or three months, until the infant can postpone his immediate satisfactions and thus fit a schedule more easily. Nevertheless, *certain urban mothers experience great difficulty in adjusting their own schedules and feelings to such a less predictable framework.* Such mothers should not be too forcefully urged to adopt the more flexible practices. Techniques of this kind must be geared to the needs of the parent as well as of the child. The emotional quality of the parent-child relationship remains more basic to healthy emotional development than any of the individual child-rearing techniques, even though these would seem to have significant values when comfortably used. The same caution is due in regard to breast feeding and the use of pacifiers,[29] which now are recognized again to have their place in infant care, as are other techniques such as rooming-in.[12] Finally, the constructive use of these more permissive techniques should not obscure the continuing need of older infants and young children for some limits on their behavior, oral or otherwise—limits geared to their developmental capacities and needs.

SUMMARY

In summary, it can be said that the psychological and psychophysiologic aspects of oral activities are importantly involved in healthy personality development, most particularly in relation to the satisfaction of the emotional needs of the infant and young child. Fortunately, later change is possible in unhealthy trends which may emerge from disturbances during these early experiences. The talisman of today is the concept of prevention, however, and the practicing physician can contribute importantly toward preventive goals.

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260 Crittenden Boulevard
Rochester 20, New York

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